



ATLAS OF POULTRY DISEASES

GUIDLINES FOR VETERINARIANS

DR. YAHIA Y. HASSAN



ATLAS OF POULTRY DISEASES

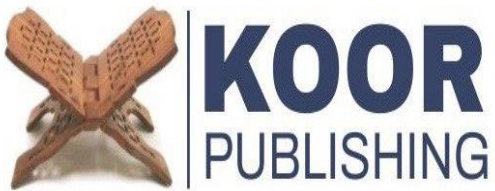
Guidelines for veterinarians

YAHIA Y. HASSAN

Address: Kebele :18, Zone 03, block 01, HN: 003,
Jigjiga, Somali Regional State, Ethiopia.
Mobile: 00251-915-74-85-34 / 00251-9-38891208
E-mail: Yaxye24@gmail.com



Published by



Books Publisher & Distributor

Jigjiga, Somali Regional State, Ethiopia.

Email: Yaxye24@gmail.com, Phone: 00251-915-74-85-34

Copyright © 2024 Yahia Y. Hassan

All rights reserved. No part of this book may be reproduced, stored in a retrieval system or transmitted in any form or by any means without the prior written permission of the publisher or in accordance with the provisions of the civil code of Ethiopia: Proclamation No.165 of 1960, Title XI. Literary and Artistic Ownership, Articles 1647 through 1974.

Photo Credits: All photos were taken by Yahia Y. Hassan, except: Fig.26, Fig.27, Fig.161, Fig.162: credited to Dr. Abdirahman D. Ibrahim and Fig.115: credited to Dr. Muhiyadin Abdilahi.

First Edition 2024

Book Cover Design by Yahia Y. Hassan

Cover Photos: Top -a hybrid of Koekoek (hen) and Indigenous chickens (cock). Bottom left: Local chicken with Infectious coryza. Bottom center: Aspergillosis; Lung shows the presence of cream color nodules. Bottom right: Bovans Brown hen with Infectious coryza.

This book is dedicated to all the poultry health professionals, who strive tirelessly to safeguard the health and well-being of poultry populations worldwide.

ACKNOWLEDGMENTS

I extend my sincerest thanks to Dr. Abdimalik Hussein, the Chief executive officer of the Somali Regional Veterinary Association (SORVA), for his pivotal visit to my home library one night while I was Writing poultry diseases in Somali language. Dr. Abdimalik Hussein's insightful comment to write the poultry diseases in English, emphasizing its potential for greater influence, sparked the inspiration that led to the creation of this "Atlas of Poultry Diseases."

I am deeply grateful for the collaborative effort and support that have contributed to the development of this "Atlas of Poultry Diseases." I extend my sincere appreciation to poultry farm practitioners; Dr. Abdirahman D. Ibrahim, Dr. Muhiyadin Abdilahi, Dr. Mintesnot Mingestu (Mintes) and Dr. Badri Nuur who have played a role in making this project a reality.

I am deeply indebted to Dr. Aydid Abdulaahi for his invaluable contributions and willingness to share his knowledge about poultry vaccination.

I extend my heartfelt gratitude to the poultry farmers, particularly grateful to Abdifatah Mahamed Mawlid whose contributions have been invaluable to the development of this "Atlas of Poultry Diseases."

Finally, I would like to thank my dear classmates and colleague, Dr. Ahmed Abdiqadir and Dr. Mubarik Abdi Yusuf (Mubarik Giire).

-----**Dr. Yahia Y. Hassan**

(D. Pharma, DVM, MSc, Assistant Professor)

PREACE

In recent years, the poultry industry in the Somali Regional State of Ethiopia has shown considerable growth and development, with an increasing number of small-scale and commercial poultry farms being established. These farms primarily focus on raising chickens for egg production, meeting the high demand for poultry products in the region.

Despite the growth and potential of the poultry industry in the region, there are significant challenges posed by various poultry diseases that commonly affect poultry farms, these diseases can have devastating effects on poultry populations, leading to significant economic losses for farmers. Recognizing the critical need to support poultry farmers, veterinarians, and industry professionals in managing these diseases effectively, the "Atlas of Poultry Diseases" was conceptualized.

Atlas of Poultry Diseases is a comprehensive visual guide that illustrates various diseases commonly affecting poultry. This resource provides detailed information on the symptoms, diagnosis, treatment, postmortem examination, prevention and control of poultry diseases, making it an essential reference for poultry farmers, veterinarians, and anyone involved in the poultry industry. By using visual aids such as photographs, and illustrations, the atlas aims to facilitate the identification and management of poultry diseases, ultimately promoting better health and welfare practices in poultry farming. This atlas contains **210 colour** photographs demonstrating the overall pathology of poultry, all photos were taken and edited (background has been changed) by Author of this book except five photos.

It is my hope that the "Atlas of Poultry Diseases" will serve as a trusted companion to all those engaged in poultry production, veterinary medicine, and academia, guiding them towards healthier flocks and a more sustainable poultry industry.

-----Dr. Yahia Y. Hassan

(D. Pharma, DVM, MSc, Assistant Professor)

TABLE OF CONTENTS

ACKNOWLEDGMENTS	iv
PREACE	v
TABLE OF CONTENTS.....	vi
DISEASES CAUSED BY VIRUS	1
Newcastle Disease.....	1
Marek’s Disease	7
Infectious Bronchitis	11
Fowl Pox	16
Infectious Bursal Disease	24
Avian Encephalomyelitis.....	27
Lymphoid Leukosis	30
Infectious Laryngotracheitis.....	32
DISEASES CAUSED BY BACTERIA	35
Infectious Coryza	35
Colibacillosis	41
Gangrenous Dermatitis.....	44
Fowl Cholera.....	47
Fowl Typhoid	50
Pullorum Disease.....	53
Necrotic Enteritis.....	56
Staphylococcosis	60
DISEASES CAUSED BY PROTOZOA	63
Coccidiosis	63

DISEASES CAUSED BY FUNGUS.....	70
Mycotoxycosis	70
Candidiasis	73
Aspergillosis.....	76
Dermatomycosis.....	80
DISEASES CAUSED BY ENDOPARASITES.....	82
Gastrointestinal Parasitism.....	82
DISEASES CAUSED BY ECTOPARASITES.....	85
Scaly Leg Mite Infestation	85
Echidnophaga Gallinacea.....	89
NUTRITIONAL DISEASES.....	95
Rickets.....	95
MISCELLANEOUS CONDITIONS.....	99
Cannibalism.....	99
Vent Gleet	103
Prolapsed Vent.....	105
Feather Loss	107
SOMALI INDIGENOUS CHICKENS	115
REFERENCES.....	118
APPENDIX.....	119
INDEX.....	126

DISEASES CAUSED BY VIRUS

Newcastle Disease

Newcastle disease (ND) is a highly contagious and often severe disease found worldwide that affects birds including domestic poultry. Chickens are particularly susceptible, morbidity and mortality rates close to 100%. Newcastle disease is the major constraint of poultry production in Ethiopia. Local names of the disease is 'Fengle' and 'Qoor-maroojiye'. The first documented evidence of Newcastle disease in Ethiopia dates back to 1974 from exotic chickens.

Etiology

Newcastle disease is caused by viruses in the serotype avian paramyxovirus type 1 (APMV-1). These viruses, which are called either APMV-1 or Newcastle disease viruses (NDV), are members of the genus *Avulavirus* in the family *Paramyxoviridae*.

Transmission

APMV-1 can be transmitted by inhalation or ingestion (fecal/ oral route). Newcastle disease is transmitted most often by direct contact with diseased or carrier birds, infected birds may shed the virus in their feces, contaminating the environment. transmission can then occur by direct contact with feces and respiratory discharges or by contaminated food, water, equipment, and human clothing. Newcastle disease viruses can survive for several weeks in the environment, especially in cool weather.

Clinical symptoms

Clinical signs are extremely variable depending on the strain of virus, species and age of bird, concurrent disease, and preexisting immunity.

Four broad clinical syndromes: These are:

1. Viscerotropic velogenic

High mortality (>90%) in susceptible flocks, inappetance (anorexia), respiratory distress (sometimes birds make a whistling sound), clear mucus discharge from the mouth, sharp drop in egg production, sudden death within 24-48 hrs, deaths continue for 7 to 10 days, profuse diarrhoea and facial edema.

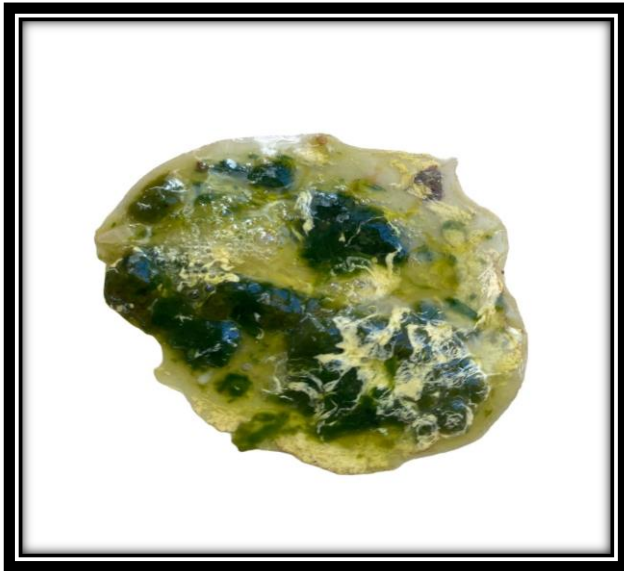


Fig.1. Newcastle disease
Greenish-white mucoid diarrhoea.



Fig.2. Newcastle disease
Facial edema.

2. Neurotropic velogenic

Mortality rate in adults 10–20%, may be much higher in young chickens, circling with clonic spasms, acute respiratory and nervous signs predominate, gasping and nervous signs: head tremors, torticollis, wing and leg paralysis.



Fig.3. Newcastle disease
Wry neck (Crookneck),
Koekoek.



Fig.4. Newcastle disease
Paralysis of legs, pullet.



Fig.5. Newcastle disease
Drooped wing, Indigenous chicken.



Fig.6. Newcastle disease
High mortality (>90%).



Fig.7. Newcastle disease
Torticollis, Pigeon.
Neck twisted 180 degrees,
head upside down.



Fig.8. Newcastle disease
Torticollis, Pigeon.
Neck curved sideways.

3. Mesogenic

Respiratory signs: gasping, nasal discharge, coughing and nerve system involvement (0-25%), drop in egg production, higher mortality (up to 50%), mild airsacculitis, tracheitis and conjunctivitis.

4. Lentogenic

Mild signs, subclinical like: mild respiratory signs, temporary loss of appetite, drop in egg production and no nervous sign, negligible mortality unless concurrent disease is present.

Postmortem lesions

Pinpoint haemorrhages on the tips of glands in the proventriculus, enlarged and haemorrhagic caecal tonsils, haemorrhagic lesions in the intestinal wall. marked congestion of trachea, often with haemorrhages. The airsacs may be inflamed (airsacculitis) and appear cloudy and congested. Airsacs may even contain cheesy (caseous) material.



Fig.9. Newcastle disease
Foamy nasal discharge,
accumulation of liquid in the lungs.

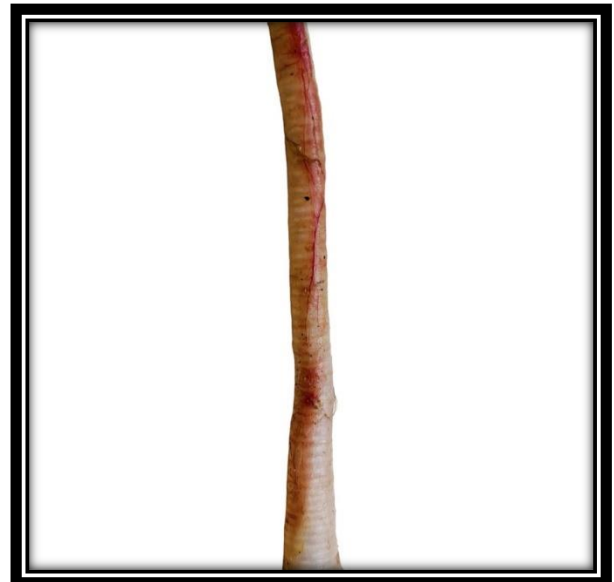


Fig.10. Newcastle disease
Marked congestion of trachea.



Fig.11. Newcastle disease
Pinpoint haemorrhages in the proventriculus.



Fig.12. Newcastle disease
Pinpoint haemorrhages in the small intestinal.



Fig.13. Newcastle disease
Pinpoint haemorrhages in the small intestinal.



Fig.14. Newcastle disease
Close up picture of the pinpoint haemorrhages small intestinal.



Fig.15. Newcastle disease
Pinpoint haemorrhages in the small intestinal, Koekoek.



Fig.16. Newcastle disease
Haemorrhages in the gastrointestinal tract.

Diagnosis

Diagnosis of Newcastle disease typically involves laboratory testing, which may include virus isolation, serological tests, and molecular diagnostic techniques like polymerase chain reaction (PCR) to confirm the presence of the virus in affected chicken.

Differential diagnosis: Infectious bronchitis, Infectious laryngotracheitis, Infectious coryza, Mycoplasmosis, Fowl cholera and Aspergillosis.

Treatment

There is no specific treatment for Newcastle disease.

Prevention and Control

Controlling Newcastle disease involves a combination of preventive measures, biosecurity practices, and in some cases, vaccination, quarantine: control wild bird contact and culling.

Marek's Disease

Marek's disease is a highly contagious, it is a viral induced-neoplastic and immunosuppressive diseases of chickens, economically important paralytic viral disease of poultry and is seriously threatening the poultry industry of the world including Ethiopia, this disease is endemic and circulating in villages in Somali regional state of Ethiopia.

Etiology

Marek's disease is caused by an *alphaherpesvirus* known as "Marek's disease virus" (MDV) or *Gallid alphaherpesvirus 2 (GaHV-2)* The causative virus is a cell-associated DNA virus belonging to the *Herpesviridae*, genus *gamma-2*.

Transmission

Main transmission is by infected premises, where day-old chicks will become infected by the oral and inhalation through the respiratory tract is the most important route of infection. Dander from feather follicles of infected chickens can remain infectious for more than a year, young chicks are particularly susceptible to horizontal transmission, susceptibility decreases rapidly after the first few days of age.

Clinical symptoms

Infected chicken show weight loss, progressive lameness or paralysis, paralysis of wings and neck, loss of weight, grey iris or irregular pupil, vision impairment, skin around feather follicles raised and roughened, Marek's disease affects the nervous system leading to paralysis typically leading to birds with a 'hurdle jumper' appearance: one wing drop and one leg forward and other leg backward (seems running by stretching) or may exhibit some form of paralysis, blindness, ataxia and wasting away.



Fig.17. Marek's disease
Showing extension of one leg
retraction of the other.
This is typically Marek's disease.



Fig.18. Marek's disease
Blindness, Indigenous chicken.



Fig.19. Marek's disease
Skin around feather follicles raised.



Fig.20. Marek's disease
Showing extension of one leg
retraction of the other in local Rooster.

Postmortem lesions

Grey-white foci of neoplastic tissue in liver, spleen, kidney, lung, gonads, heart, and skeletal muscle, diffuse, blotchy haemorrhage in proventriculus and enlargement of a sciatic nerve.



Fig.21. Marek's disease
Liver showing white spots of cancerous tissue on their surface.
In commercial layer, Bovans Brown.



Fig.22. Marek's disease
Diffuse, blotchy haemorrhages in proventriculus.



Fig.23. Marek's disease
Enlargement of a sciatic nerve, Dekalb White pullet.

Diagnosis

History, gross lesions and histopathology, serology: (ELISA) and molecular methods, such as PCR, can be used to detect carrier birds.

Differential diagnosis: Lymphoid leukosis, Botulism, Deficiency of thiamine, Deficiency of Ca/Phosphorus/Vitamin D, especially at the start of lay.

Treatment

There is no effective treatment for Marek's disease.

Prevention and Control

Strict biosecurity measures, use of genetically resistant chicks, vaccination of day-old chicks is an effective mean of control and requests for vaccinations should be on chicks purchased from hatcheries.

Infectious Bronchitis

Infectious bronchitis is an acute and highly contagious respiratory disease of chickens and economically important disease. Infectious bronchitis is present worldwide, Infectious bronchitis virus are widespread in unvaccinated backyard chicken and commercial farms in Somali regional state of Ethiopia. birds of all ages are susceptible but the disease is most common in young chicks, causing high mortality.

Etiology

Infectious bronchitis (IB) is caused by avian infectious bronchitis virus a coronavirus (*Coronaviridae*, *Orthocoronavirinae*, genus *Gammacoronavirus*, subgenus *Igacovirus*).

Transmission

The virus is transmitted rapidly from bird to bird through the airborne route, the virus can also be transmitted via the air between chicken houses and even from farm to farm.

Clinical symptoms

1. **Respiratory form:** Is the most common in birds of all ages, typical respiratory signs such as coughing, sneezing, rales, wheezing, and gasping are present, watery discharge from the eyes and/or nostrils, swelling of the head, and head shaking may be observed, feed and water consumption decline, breathing noises are more noticeable at night while the birds rest. Chicks are weak, have a loss of appetite, reduced water intake, depression, and huddle around heat sources.
2. **Reproductive form:** Egg producing birds may have severe and prolonged reduction in egg laying; the few eggs laid have soft or rough shells, and the egg white becomes watery, production recovers in 5 or 6 weeks, broken eggs in the abdomen may occur and result in inflammation of the peritoneum (egg yolk peritonitis).
3. **Kidney form:** Swollen kidneys, with distended ureters and tubules containing whitish uric acid crystals.



Fig.24. Infectious bronchitis
Sudden onset of discharge from eye of a chicken. This may be of Infectious bronchitis or Infectious coryza.



Fig.25. Infectious bronchitis
The symptom of severe respiratory Distress, Bovans Brown.



Fig.26. Infectious bronchitis
Shell-less egg.
Courtesy of Dr. Abdirahman D. Ibrahim



Fig.27. Infectious bronchitis
Marked drop in egg production and increased number of poor quality eggs-soft shelled with watery content.
Courtesy of Dr. Abdirahman D. Ibrahim

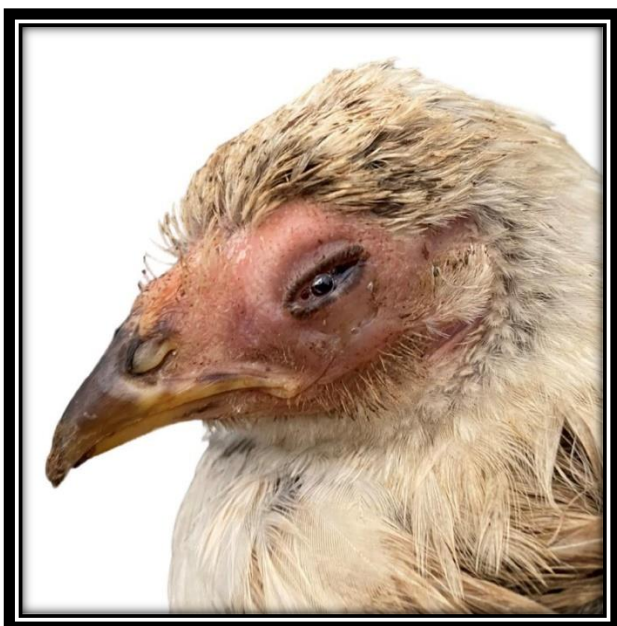


Fig.28. Infectious bronchitis
Sudden onset of discharge from eye of local chick and swelling of the head.



Fig.29. Infectious bronchitis
Respiratory signs: difficulty in breathing (open beak).

Postmortem lesions

young chicks a yellow cheesy plug at the tracheal bifurcation is indicative of IB infection. In case of nephropathogenic infections pale and swollen kidneys and distended ureters with urates are found in older bird mucus and redness in the trachea, exudate in the air sacs, and various changes in the oviduct depending on the time and severity of infection. In case of “false layers” permanent lesions in the oviduct make egg production impossible. The oviduct will be blocked and filled with fluid (cysts) or never developed into an active oviduct.



Fig.30. **Infectious bronchitis**
False layer syndrome.



Fig.31. **Infectious bronchitis**
Tracheitis with mucus deposit in commercial layer. (close up view)



Fig.32. **Infectious bronchitis**
Cystic oviduct in an affected (false) layer.

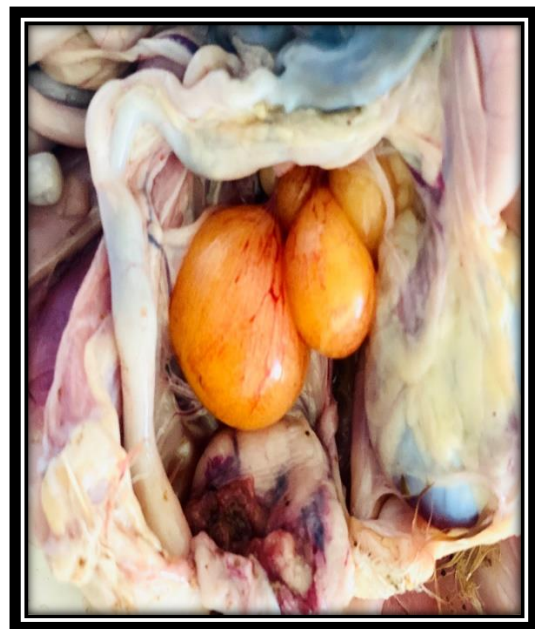


Fig.33. **Infectious bronchitis**
Salpingitis.

Diagnosis

A tentative diagnosis is based on clinical signs and lesions, there are three main factors to be considered in order to arrive at a diagnosis.

1. The clinical picture including post-mortem findings in the flock.
2. Isolation of the virus in the laboratory.
3. A rising antibody titre when the serum is tested against a known strain of bronchitis virus, ELISA.

Differential diagnosis: Newcastle disease and Infectious laryngotracheitis.

Treatment

There is no treatment for Infectious bronchitis.

Prevention and Control

Mortality can be reduced by supportive treatment: antibiotics are used to control secondary bacterial infections and elimination of overcrowding, strict biosecurity and prevention of stress (cold stress). Vaccination with strain specific or cross protective live vaccines, and for layers and breeders the addition of inactivated vaccines at point of lay to induce long lasting systemic immunity.

Fowl Pox (Dhow)

Fowl pox, also known as Avian Pox is slowly spreading viral disease characterized by skin lesions and/or plaque in the pharynx and affecting chickens, turkeys, pigeons and canaries worldwide. It is endemic in poultry in Somali regional state of Ethiopia. Local names of the disease is '**Dhow**' Or '**Furuqa shimbiraha**'. The disease is mainly observed in commercial farms and local chickens kept under a scavenging system.

Etiology

The etiologic agent of Fowl pox is the fowlpox virus, the type virus of the genus *Avipoxvirus* in the family Poxviridae.

Transmission

Fowl pox is transmitted to flocks primarily through biting mosquitoes (*Culex* and *Aedes* species), or new birds introduced to your flock may be latent carriers of the disease, spread of the virus from one bird to another by direct contact is the method of spread. Most of the spread is the result of birds picking, fighting, or scratching one another.

Clinical symptoms

The incubation period varies from 4 to 20 days, affected birds will be depressed, weakness inappetence, poor growth, poor egg production. Mortality is variable, from a low 1 to 2%, when slight head lesions are present, to over 40% when the diphtheritic form ("wet pox") is more prevalent. There are two forms of the disease:

1. **Cutaneous form (dry form):** The cutaneous or dry form is the most common, is characterized by nodular lesions on various parts of the un-feathered skin of the head, neck, comb, wattles, eyelids, leg and feet. The lesions on the head, combs, and wattles are usually wart-like (nodular) in appearance, and yellow to dark brown in colour.
2. **Diphtheria form (wet form):** Raised-yellow white cheesy patches or Cankers are imbedded in the membranes of the mouth, larynx and trachea this causes bird dies suddenly from asphyxiation (lack of oxygen).

1. Chicks

Chicks are highly susceptible to Fowl pox.



Fig.34. **Fowl pox**
Wart –like nodules on chick,
Koekoek chick.



Fig.35. **Fowl pox**
Wart –like nodules on chick
depressed, Indigenous chick.



Fig.36. **Fowl pox**
Scab formation around corner of the
mouth, Indigenous chick.



Fig.37. **Fowl pox**
Scab formation on eye,
Indigenous chick.

2. Early-stages of fowl pox (dry form)

The lesions begin as small blisters and then progress into wart-like nodules.



Fig.38. **Fowl pox**
Pox lesion on face chicken.



Fig.39. **Fowl pox**
Pox lesion around eye chicken.



Fig.40. **Fowl pox**
Small scab formation
around face chicken.



Fig.41. **Fowl pox**
Nodular in appearance
and yellow in colour.

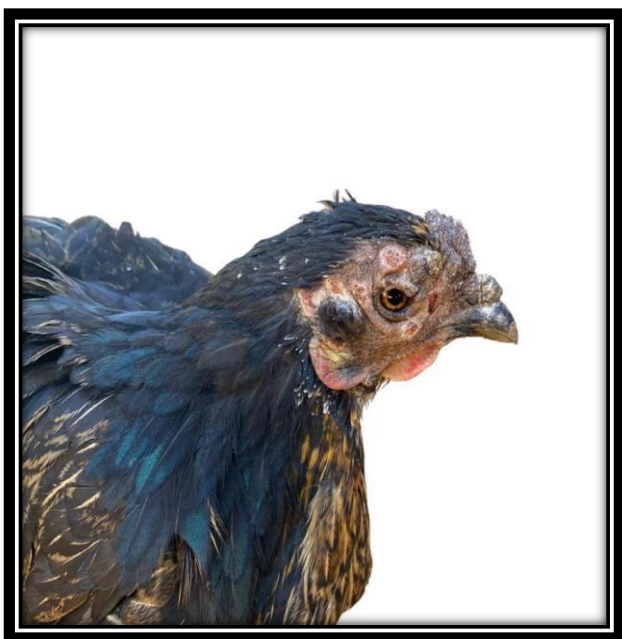


Fig.42. **Fowl pox**
Scab-like lesion, laying hen.



Fig.43. **Fowl pox**
Wart-like nodules on the face.



Fig.44. **Fowl pox**
Scab-like lesion,
local breeder, male



Fig.45. **Fowl pox**
Scab formation around eye.

3. Late-stage of fowl pox (dry form)

As a result of discharge or scab formation, the eyelids may become sealed shut, the chickens could potentially die of starvation.



Fig.46. **Fowl pox**
The eyelids may become sealed shut.

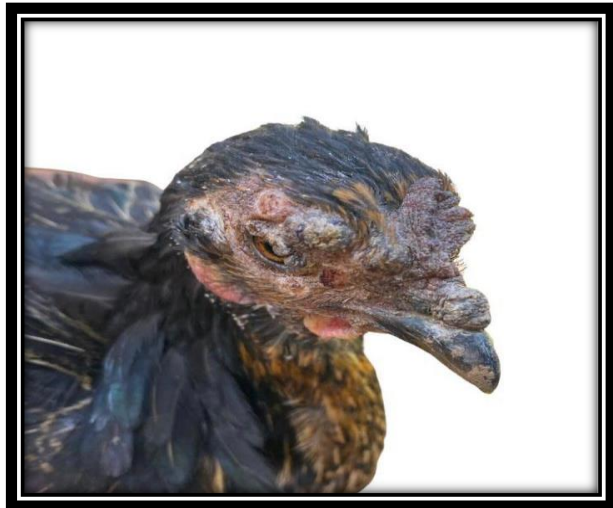


Fig.47. **Fowl pox**
Scab formation around nostrils
blockage, asphyxiation.



Fig.48. **Fowl pox**
The eyelids may become sealed shut.



Fig.49. **Fowl pox**
Eventually die due to starvation or
asphyxiation

4. Pigeons

There is a potential risk that they can transmit Fowl pox to chickens if they come into contact with infected birds.



Fig.50. Fowl pox
Pigeon infected by Fowl pox
(diphtheritic form).



Fig.51. Fowl pox
Scab formation around beak, Pigeon.

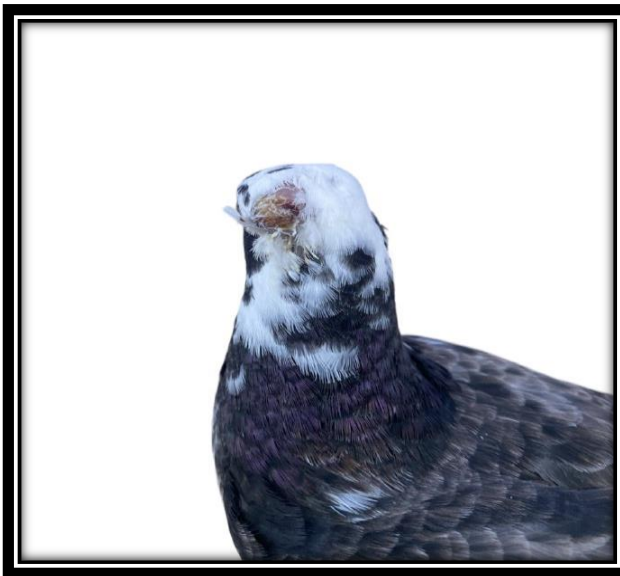


Fig.52. Fowl pox
Scab formation on head, Pigeon.



Fig.53. Fowl pox
Scab formation on beak, Pigeon.



Fig.54. **Fowl pox**
White discharge.



Fig.55. **Fowl pox**
Pigeon infected by Fowl pox
(diphtheritic form).



Fig.56. **Fowl pox**
Pox lesion on body of young pigeon.

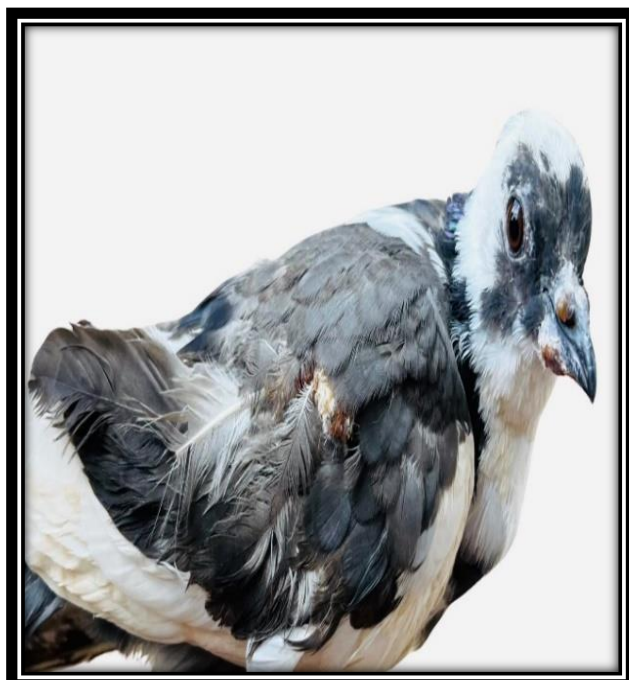


Fig.57. **Fowl pox**
Scab formed on wing of pigeon.

Postmortem lesions

Papules progressing to vesicles then pustules and scabs, histological examination shows characteristic intracytoplasmic inclusion bodies in infected skin and tracheal mucosa. The diphtheritic form is recognized by the presence of nodular hyperplasia of the mucosa of the pharynx and trachea. Chickens which die of diphtheritic pox may show a plug of desquamated epithelium which lodge in the glottis resulting in asphyxiation.

Diagnosis

Usually diagnosed by clinical signs, characteristic gross and microscopic lesions. Microscopic examination of affected tissues stained with H&E reveals eosinophilic cytoplasmic inclusion bodies. This is the most commonly used method in diagnostic laboratories. PCR assay for detection of the fowl pox virus-specific genes.

Differential diagnosis: Vitamin A deficiency and Infectious laryngotracheitis.

Treatment

There is no effective treatment.

Prevention and Control

Preventive and control strategies include reducing the local insect population, reducing skin trauma from fighting, and vaccination in endemic areas, chickens may be vaccinated at day 72 of age using the wing web-stick method and supportive care and appropriate antibiotic therapy may provide protection from secondary bacterial complications.

Recovery: Most individuals with fowl pox will recover and will clear the virus, recovered birds are not reservoirs of infection. Flocks can recover from infection, but the environment must be cleaned and vectors controlled before new birds are introduced.

Infectious Bursal Disease

Infectious bursal disease (IBD), also known as Gumboro disease is a highly contagious disease of young chickens and turkeys, characterized by immunosuppression and mortality generally at 3 to 6 weeks of age. The disease was first discovered in Gumboro, Delaware in 1962. It is economically important to the poultry industry worldwide. Infectious bursal disease has become a priority problem in commercial and backyard poultry production systems in Somali Regional State of Ethiopia.

Etiology

The etiologic agent of infectious bursal disease is the infectious bursal disease virus (IBDV), a double-stranded RNA virus (*Avibirnavirus gumboroense*) in the family Birnaviridae. As with other birnaviruses, IBDV virions are nonenveloped and have an icosahedral single-shelled geometry.

Transmission

Infectious bursal disease virus is very infectious and spreads easily from bird to bird by way of droppings, infected clothing and equipment are means of transmission between farms, The route of infection is usually oral, but may be via the conjunctiva or respiratory tract. There is no vertical transmission, the virus is very resistant, persisting for months in houses and faeces.

Clinical symptoms

Incubation period of infectious bursal disease is very short and clinical signs occurs within 2-3 days of exposure, sudden drop in feed and water consumption, tendency for some birds to pick at their own vents. Other symptoms include depression, white watery droppings leading to soiling of feathers around the vent, loss of appetite, ruffled feathers, unwillingness to move, trembling, closed eyes.



Fig.58. Infectious bursal disease
Young chicken is depressed, ruffled
feathers and closed eyes, Bovans Brown.



Fig.59. Infectious bursal disease
White milky running stool.

Postmortem lesions

Post mortem of dead birds reveals the following lesion, muscular haemorrhages in breast & thigh, bursa enlarged or atrophied, haemorrhagic, mucus, gelatinous exudates or cheesy mass, haemorrhages between proventriculus & gizzard junction and swollen kidneys with urates.

Diagnosis

Typical clinical signs and post mortem lesions are found after IBD infection. both serology (ELISA) and molecular methods can diagnose infection in a flock.

Differential diagnosis: Sulfonamide poisoning, Aflatoxicosis, and Pale bird syndrome (Vitamin E deficiency).

Treatment

There is no specific treatment.

Prevention and Control

Vaccination of breeders and young chicks is the best means of control, use of multivitamin supplement and facilitating access to water may help, antibiotic medication may be indicated if secondary bacterial infection occurs, depopulation and rigorous of contaminated farms, strict hygienic measures including control of personal and material movement and application of All-in /All-out procedures.

Avian Encephalomyelitis

Avian encephalomyelitis, also known as epidemic tremor is a viral disease of the central nervous system of chicks. Avian encephalomyelitis (AE) occurs world-wide, resulting in an asymptomatic egg production decline in commercial layers and breeders and elevated mortality in vertically infected batches of chicks.

Etiology

The etiological agent of Avian encephalomyelitis is avian encephalomyelitis virus (AEV), a nonenveloped RNA virus (family Picornaviridae, genus *Tremovirus*, species *tremovirus A*). Natural field strains of the virus are enterotropic and multiply in the intestine. Infected birds shed the virus in their feces for a few days to a few weeks, which serves to transmit the infection to hatchmates.

Transmission

The transmission of the virus is both vertical and horizontal, vertical transmission tends to cause higher losses because day-old chicken are affected. This happens when breeders with no immunity are infected during the egg production period and transmit the virus to progeny. This virus can also be transmitted horizontally by fecal-oral route through ingestion of feed, litter, or contaminated water.

Clinical symptoms

Young chicks

The main clinical signs of Avian encephalomyelitis in young chicks are depression, muscular incoordination, ataxia and leg weakness that varies from sitting on hocks to paresis that progresses to paralysis and recumbency, fine tremors of the head and neck are evident in some birds and are characteristic of the disease, they are responsible for the common name epidemic tremor (Gariirka safmarka).

laying and breeding flocks

Avian encephalomyelitis virus infection causes a marked drop in egg production, chickens which survive the clinical disease may develop cataracts later in life.

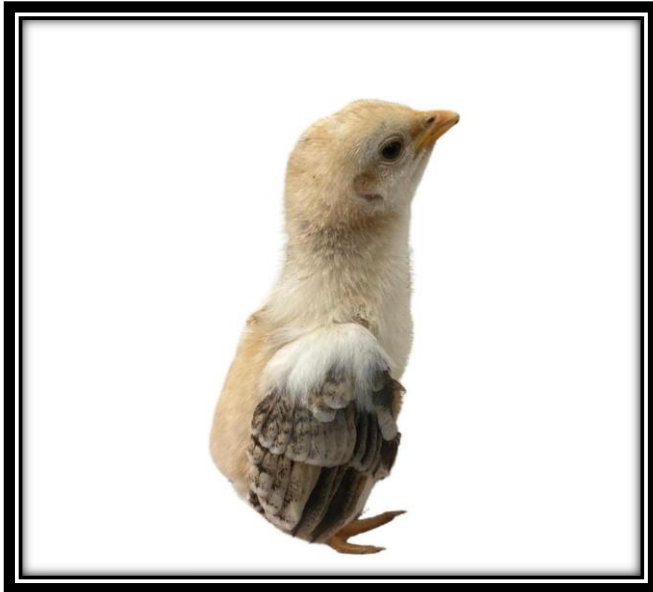


Fig.60. **Avian encephalomyelitis**
Chick affected by Avian encephalomyelitis,
sitting on hocks.



Fig.61. **Avian encephalomyelitis**
Chick presenting neurological signs of
Avian encephalomyelitis.

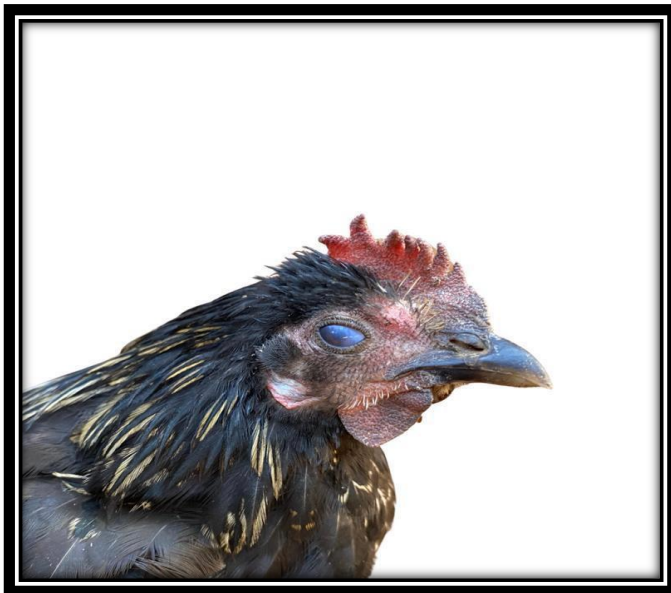


Fig.62. **Avian encephalomyelitis**
Opacity of eye lenses (cataracts) in
old chicken after infection.

Postmortem lesions

No gross lesions are observed.

Diagnosis

Presumptive diagnosis is based on clinical signs presented in chicks, together with a drop in production and hatchability in the parent breeders, is indicative of Avian encephalomyelitis infection. Serology: commercial ELISA test kits are available to monitor the antibody titer of flocks to determine susceptibility or following vaccination or challenge.

Differential diagnosis: Vitamin E deficiency, Newcastle disease (neurologic form) and Marek's disease.

Treatment

There is no specific treatment for Avian encephalomyelitis.

Prevention and Control

Preventive vaccination of breeder pullets with live Avian encephalomyelitis vaccine before egg production is the only effective means of control Avian encephalomyelitis.

Lymphoid Leukosis

Lymphoid leukosis, also known as big liver disease is the commonest neoplasm in chicken caused by avian leukosis virus (ALV). It usually occurs between 14th to 30th week of age. Incidence is highest at about sexual maturity, Lymphoid leukosis is usually a problem of laying hens. The disease can be immunosuppressant which increases susceptibility to other diseases.

Etiology

Lymphoid leukosis virus is an RNA virus in the family of retroviruses. Ten viral subgroups (A-J) are known. Avian leukosis viruses (ALVs).

Transmission

Lymphoid leukosis virus is transmitted both vertically from hen to progeny through the egg, and also horizontally from bird to bird by direct or indirect contact. Usually only a small number of chicks are infected vertically, and the majority become infected by contact with vertically infected chicks during rearing.

Clinical symptoms

Signs of Lymphoid leukosis begin to show in birds only after the age of 16 weeks, because it has a long incubation period. Clinical signs are non-specific. weight loss, depression, paleness, emaciation, weakness, inappetance. Feed/gain ratio increased, decreased egg production and increased culls, external tumours may be seen and greenish diarrhea develops in terminal stages.

Postmortem lesions

Post-mortem examination reveals organ enlargement, particularly in the liver and spleen, due to the presence of tumors, tumors may also develop in the kidney, lungs, ovaries, testicles, and bone marrow, the size of the tumors can vary, and they are typically soft in consistency.

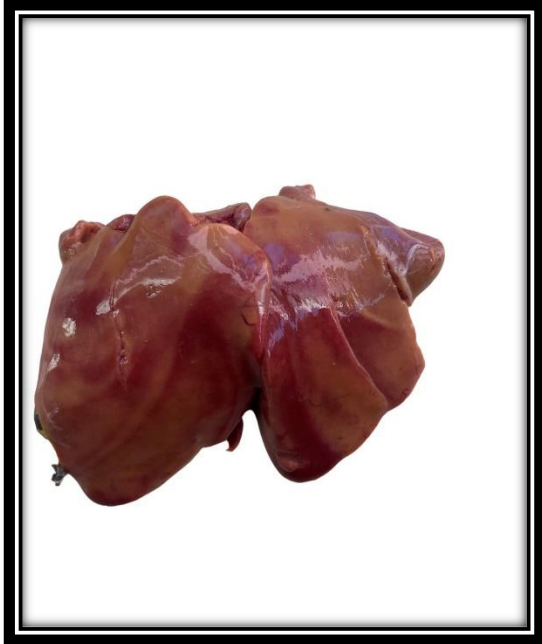


Fig.63. lymphoid leukosis
Marked enlargement of the liver.
big liver.

Diagnosis

Standard criteria: history and clinical signs, gross pathology, and histopathology Advanced criteria: immunohistochemistry, standard and quantitative PCR, virus isolation, serology.

Differential diagnosis: Marek's disease, when distinct lymphoid tumours are present in the bursa, a diagnosis of Lymphoid leukosis can be made. Such tumours in bursa are absent in Marek's disease.

Treatment

There is no treatment for Lymphoid leukosis.

Prevention and Control

Prevention is also helped by obtaining chicks from breeder flocks that are free of the virus and rearing birds in isolation with adequate ventilation. Follow good biosecurity and management procedures to prevent stress and control other diseases, and dispose of dead birds.

Infectious Laryngotracheitis

Infectious laryngotracheitis (ILT) is an acute, highly contagious herpesvirus infection of chickens, pheasants, peafowl and turkeys. The effects of this disease are largely commercial as it can cause deaths and affects productivity.

Etiology

Infectious laryngotracheitis caused by the infectious laryngotracheitis virus, also known as Gallid herpesvirus 1 (GaHV-1) belonging to the genus *Iltovirus*, subfamily *Alphaherpesvirinae*

With in *Herpesviridae* family.

Transmission

Bird to bird by the respiratory route, most outbreaks of Infectious laryngotracheitis on farms are traced back to transmission by contaminated people or equipment (visitors, shoes, clothing, egg boxes, used feeders, waterer, cages, crates etc.).

Clinical symptoms

Clinically, the disease may appear in three forms:

1. Peracute form

Onset of disease is sudden with a rapid spread, the morbidity is high and mortality may exceed 50%. Some birds may die in good body condition before the appearance of signs, gasping, coughing bloody mucoid exudate, rattling, and extension of the neck during inspiration are seen 5–12 days after natural exposures.

2. Subacute form

The onset of illness is slower and respiratory signs may extent over some days before deaths are seen, the morbidity is high but the mortality is lower than in the peracute form.

3. Chronic or mild form

May be seen among survivors of either of the above forms of disease, although some out breaks themselves may be entirely mild. Sign: conjunctivitis, sinusitis, oculo-nasal discharge, respiratory distress, swollen orbital sinuses and decreased egg production.

Postmortem lesions

Lesions are found throughout the respiratory tract but most pronounced in the larynx and trachea. Depending on the severity of the infection you can find tracheitis with haemorrhagic and/or diphtheric changes.



Fig.64. Infectious laryngotracheitis
Mild congestion of the trachea.



Fig.65. Infectious laryngotracheitis
Haemorrhages in trachea.



Fig.66. Infectious laryngotracheitis
Blood in the lumen of the trachea in acute form of ILT (close-up view).

Diagnosis

Infectious laryngotracheitis in chicken can be tentatively diagnosed based on the clinical signs such as conjunctivitis, gasping, open mouth or extended head respiration, expectoration of bloody. In the laboratory a definite diagnosis can be made by histological examination of tracheal tissues or virus isolation from tracheal mucus in embryonated chicken eggs.

Differential diagnosis: Newcastle disease and Infectious bronchitis

Treatment

There is no treatment.

Prevention and Control

Good biosecurity practices combined with vaccination are the practical methods to control ILTV, other Methods of prevention include strict biosecurity, quarantine or all-in/all-out operation. antibiotics can be used to control secondary bacterial infection if this is marked.

DISEASES CAUSED BY BACTERIA

Infectious Coryza

Infectious coryza (IC), also known as 'fowl coryza' is an acute and contagious respiratory disease of chickens, chickens of all ages are susceptible and the susceptibility increases with age and older birds suffer more severely. Infectious coryza is a common problem in the Somali region of Ethiopia, results in decreased egg production in commercial laying and indigenous chickens. Usually the disease shows high morbidity and a low mortality.

Etiology

The causative bacterium of infectious coryza is *Avibacterium paragallinarum*, a gram-negative, pleomorphic, nonmotile, catalase-negative.

Transmission

Infectious coryza is transmitted from bird to bird through direct contact, and via contaminated feed and water, airborne droplets, Birds which have recovered from the disease remain carriers and chronically ill or healthy carrier birds are the reservoir of infection for *A. paragallinarum*.

Clinical symptoms

Infectious coryza has a short incubation period that develops clinical signs within 1-2 days after inoculation in to the chickens, the most prominent Clinical signs of Infectious coryza are an acute inflammation of the upper respiratory tract including involvement of nasal discharge and sinuses with a serous to mucoid nasal discharge, sneezing, facial edema and swelling of the face under eye, conjunctivitis, anorexia, and retarded growth in young poultry.

In addition to this, there may be diarrhea, decrease in feed and water consumption and a reduction in egg production in layer chickens, losses due to persistent mortality are culling where up to 5%. Affected flocks may suffer an egg drop of up to 10%, and to 100% in more serious situations.



Fig.67. Infectious coryza
Sudden onset of discharge from eye of pullet, Bovans Brown.



Fig.68. Infectious coryza
Sudden onset of discharge from eye of a layer, Bovans Brown.



Fig.69. Infectious coryza
Hen with infectious coryza exhibiting periorbital edema, swollen infraorbital sinus and nasal exudate.



Fig.70. Infectious coryza
Conjunctivitis in local chicken.



Fig.71. Infectious coryza
One or both infraorbital sinuses will continue to swell, preventing the eyes from opening completely. (Unilateral)



Fig.72. Infectious coryza
Facial edema and swelling of the face under eye.



Fig.73. Infectious coryza
Eyelids can become crusty and stuck together.



Fig.74. Infectious coryza
Swelling of the face under eye.



Fig.75. Infectious coryza
The initial inflammation is watery.
Later, it becomes thicker and white like
pus, and it has a foul odor.



Fig.76. Infectious coryza
Thicker and white like
pus in local rooster.



Fig.77. Infectious coryza
If not treated it cause eye damage.



Fig.78. Infectious coryza
Thicker and white like
pus in local chicken.



Fig.79. **Infectious coryza**
Eyelids can become crusty
and stuck together, blindness.



Fig.80. **Infectious coryza**
Eye edema.

.

.

.

Postmortem lesions

Catarrhal inflammation of nasal passages and sinuses, conjunctivitis, eye-lid adherence, caseous material in conjunctiva/sinus and tracheitis.

Diagnosis

A presumptive diagnosis may be made on signs, history, lesions and identification of the bacteria in a Gram-stained smear from sinus.

Differential diagnosis: Chronic fowl cholera, Mycoplasmosis, Laryngotracheitis, Newcastle disease, Infectious bronchitis, Avian influenza and Vitamin A deficiency.

Treatment

Doxycycline hyclate 200 mg, 250-600 g per 1000 liters of drinking water during 3-5 days (Equivalent to 20 mg per kg body weight), Or

Oxytetracycline (20%), 200g/100 liters of drinking water for 3-5 days. Or

Enrofloxacin, one liter suspension /1500-4000 liters of drinking water for 3-5 days. Or

Sulfamethazine-trimethoprim, 1000mg in drinking water or 2000g/ton for 3 days. Or

Amoxicillin 16mg/kg bw or 100g per 100 liters for 3-5 consecutive days.

Prevention and Control

The prevention and control of Infectious coryza depends on strict biosecurity, use of antiseptics, disinfectants and antibiotics, stock coryza-free birds on an all-in/all-out production policy. Live attenuated strains vaccines are used in areas of high incidence.

Colibacillosis

Colibacillosis is one of the most commonly occurring and economically devastating bacterial diseases of poultry worldwide. It is frequently associated with immunosuppressive diseases. Colibacillosis causes elevated morbidity and mortality leading to economic losses on a farm especially around the peak of egg production and throughout the late lay period.

Etiology

Colibacillosis is caused by *Escherichia coli*. *E. coli* are gram-negative, rod-shaped bacteria and are a common inhabitant of the lower bowel. Although most *E. coli* are non-pathogenic, the Avian Pathogenic *E. coli* (APEC) strains are able to establish themselves outside of the intestines and cause disease.

There are three predisposing factors

1. Diseases factor

Respiratory stress caused by infections with *Mycoplasma gallisepticum* or viral agents such as IBV, NDV, and IBD, Poor navel healing, mucosal damage due to viral infections and immunosuppression or immature immunity in chicken are predisposing factors for other forms of diseases.

2. Environmental factor

Temperature, humidity, & high concentrations of ammonia and dust in poultry house,

3. Management factor

Overcrowding, poor ventilation, housing system, poor litter condition and stress

Transmission

Infection is by the oral or inhalation routes, and via shell membranes/yolk/navel. The infectious agent is moderately resistant in the environment, but is susceptible to disinfectants and to temperatures of 80°C.

Clinical symptoms

It has an incubation period of 3-5 days, reduced appetite, poor growth, omphalitis and respiratory signs: distress, coughing and sneezing. *E. coli* can cause an infection under the skin, known as cellulitis, and is commonly associated with respiratory disease in birds, which in severe cases leads to colisepticaemia and death. It can also cause salpingitis, yolk sac infection (Egg peritonitis, mushy chick).

Postmortem lesions

Lesions vary from acute to chronic in the various forms of the disease, airsacculitis, peritonitis, enteritis, pericarditis swelling and granulomata in liver and spleen, salpingitis, synovitis, arthritis, omphalitis and cellulitis over the abdomen or in the leg.

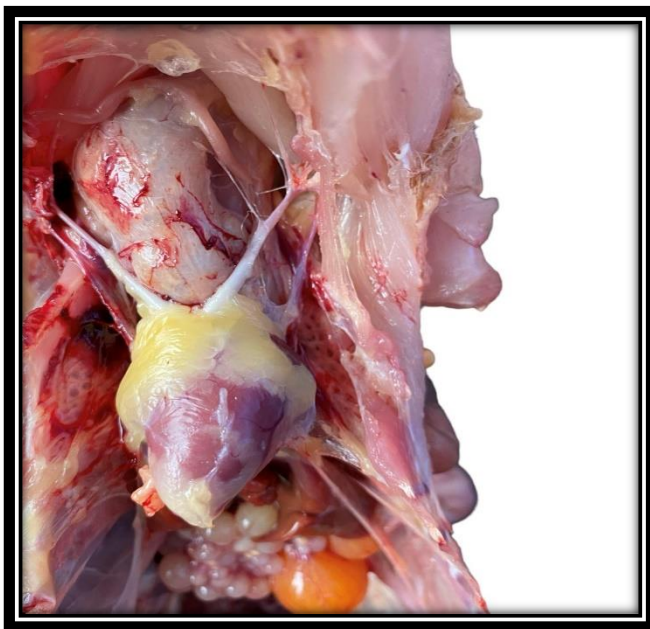


Fig.81. Colibacillosis
Pericarditis in commercial layer,
Bovans Brown.



Fig.82. Colibacillosis
Egg yolk peritonitis in
Dekalb White laying hen.

Diagnosis

Pathology, isolation and sero-typing, aerobic culture yields colonies of 2-5mm on both blood and McConkey agar after 18 hours - most strains are rapidly lactose-fermenting producing brick-red colonies on McConkey agar.

Differential diagnosis: Fowl cholera, Pullorum disease and Chronic respiratory disease.

Treatment

Chlorotetracycline HCl, 100-125 mg per kg body weight, or 100 g per 750-1000 liters of drinking water during 4-5 days. Or

Doxycycline hyclate 200 mg, 250-600 g per 1000 liters of drinking water during 3-5 days (Equivalent to 20 mg per kg body weight), Or

Sulfamethazine-trimethoprim suspension, 1ml /5 liter of drinking water daily for 5 days. Or

Amoxycillin trihydrate (20-50%) powder, 200g per 600 ml of drinking water (for 2000 birds each 1 kg) 20mg/kg, q 24 h for 3-5 (33.3%). Or

Tetracycline 13.3 gm/ 120 liters of drinking water (equivalent to 60 mg/kg feed q 24 h) for 3-5 days. Individual bird could be treated at 5% SC.

Prevention and Control

Maintain the highest standards of management and sanitation, avoid overcrowding, avoid dry, dusty conditions, provide adequate ventilation in the poultry house, avoid build-up of ammonia, maintain proper litter conditions, protect the birds against extremes of temperature, heat or cold, ensure supply of clean water. Also use water sanitizers, contaminated water can contain organisms in high numbers, to prevent yolk sac infection and early chick mortality, provide the best possible brooding condition, provide well-balanced diet to avoid mineral deficiencies and malnutrition and ensure a supply of high protein, vitamin A, vitamin E, vitamin C, and zinc.

Gangrenous Dermatitis

Gangrenous dermatitis (GD), Also known as "wing rot", is a sporadic skin infection characterized by necrosis of the skin and subcutaneous tissue of breast of poultry. It is serious bacterial disease affecting poultry. It generally is fatal, often within 24 hours. It is mainly a disease of the commercial layers, turkeys, and broiler breeders, it usually occurs in fast-growing broilers between 4 and 8 weeks of age. Its occurrence is more common in warm, humid conditions. Mortality ranges from 1 to 60%.

Etiology

The primary causes of Gangrenous dermatitis are *Clostridium septicum* and *C perfringens* type A; these microorganisms may act singly or in combination, several other anaerobic and aerobic bacteria may also cause GD. Amongst those are, *Staphylococcus aureus*, *Escherichia coli*, and *Pasteurella multocida*,

Transmission

Transmission is by contact with infected wet, caked litter. The disease often occurs in immunosuppressed birds.

Clinical symptoms

The incubation period of gangrenous dermatitis is usually less than 24 hours. Most affected birds are well-fleshed. The main clinical signs are sudden onset, depression fever, listlessness, anorexia, ataxia, and recumbency. The lower parts of the body and inner thighs frequently have an accumulation of subcutaneous edema, the skin of affected areas is usually devoid of feathers and shows dark reddish, purple, green, or green-blue discoloration, the most frequently affected areas of the body are breast, abdomen, back, thighs, legs, and wings, cases of GD may also occur without clinical signs being observed.



Fig.83. **Gangrenous dermatitis**
Skin and tissue on wing deep red,
moist and swollen,
White leghorn layer.



Fig.84. **Gangrenous dermatitis**
The skin of affected areas is intense
intense red swollen, devoid of feathers,
White leghorn layer.

Postmortem lesions

Lesions are hemorrhagic beneath the skin, reddish, greenish, necrotic skin usually devoid of feathers, overlying wings, breast, abdomen, or legs. Gas may be given off upon palpation of skin, and blood-tinged edema (watery fluid). It may affect muscle under skin causing discoloration. Anemia (paleness due to lack of red blood cells) and retained yolk sacs.

Diagnosis

Presumptive diagnosis of Gangrenous dermatitis is based on clinical signs and post-mortem lesions, which include congestion, hemorrhage and necrosis of skin, definitive diagnosis is based on isolation and identification of *Clostridium septicum* or *Staphylococcus aureus* (or both).



Fig.85. Gangrenous dermatitis
Hemorrhagic beneath the skin in a Fayoumi layer.

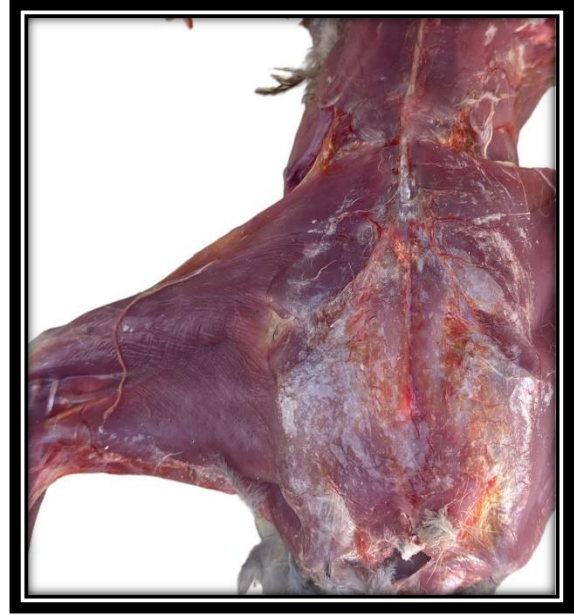


Fig.86. Gangrenous dermatitis
Extensive haemorrhagic, cellulitis of the subcutaneous tissue.

Treatment

Doxycycline hyclate 200 mg, 250-600 g per 1000 liters of drinking water during 3-5 days (Equivalent to 20 mg per kg body weight), Or

Erythromycin, 500mg/gallon of water, for 7 days. Or

Amoxicillin 16mg/kg bw or 100g per 100 liters for 3-5 consecutive days. Or

Tilmicosin (as phosphate) 25%, 300ml per 1000 liter of drinking water during 3-5 days (20mg/kg body weight)

Prevention and Control

Avoiding overcrowding, strict sanitation, cleaning and disinfection of houses, appropriate litter management, prevention of trauma, controlling cannibalism, removing dead birds regularly and vaccination against immunosuppressive viral diseases.

Fowl Cholera

Fowl cholera (FC), also known as Avian Pasteurellosis is a septicaemic disease (blood infection) of chickens, Fowl cholera is a serious, highly contagious disease affecting a range of avian species including chickens, turkeys, and Water fowl. Fowl cholera usually strikes birds older than 6 weeks of age, the disease is present globally and endemic to most parts of Somali regional state of Ethiopia and commonly reported in commercial flock with significant economic losses associated with reduced production and mortality.

Etiology

Pasteurella multocida, the causative agent of fowl cholera, is a small, gram-negative, nonmotile rod with a capsule that may exhibit pleomorphism after repeated subculture.

Transmission

The route of infection is oral or nasal via nasal exudate, faeces, contaminated soil, equipment, and people, transmission of fowl cholera is mainly from bird to bird by water or feed contamination, vectors like flies and red mite can be carriers and can add to the spread. Rodents also appear to play a role in contamination of water and feed with *Pasteurella multocida*: Rats are reservoirs for *Pasteurella multocida*.

Clinical symptoms

In acute: Outbreaks, dead birds may be the first sign. fever, reduced feed consumption, mucoid discharge from the mouth, ruffled feathers, diarrhea, and labored breathing may be seen. As the disease progresses birds lose weight, become lame from joint infections, and develop rattling noises from exudate in air passages.

In chronic: Chickens develop abscessed wattles and swollen joints and foot pads, caseous exudate may form in the sinuses around the eyes, conjunctivitis, rales and torticollis.



Fig.87. Fowl cholera
 . The periorbital sinuses are affected by a serofibrinous inflammation,
 Bovans brown.



Fig.88. Fowl cholera
 Swollen wattle in local breeder,
 Conjunctivitis (lateral view).



Fig.89. Fowl cholera
 Swollen wattle this is
 characteristic of chronic
 cases of fowl cholera.



Fig.90. Fowl cholera
 Swollen foot in Bovans Brown.

Postmortem lesions

Peracute disease produces no lesions, haemorrhage on heart and fat, conjunctivitis, subepicardial and subserosa haemorrhage, congested breast, and septicemia can occur with chronic form. free yolk in abdominal cavity, liver-yellow brown streaks, “egg yolk” peritonitis, consolidation of lungs, mucous in the mouth and nasal passages, and caseous exudate in the palatine cleft (upper part of the mouth) may also be seen.

Diagnosis

Impression smears, isolation (aerobic culture on trypticase soya or blood agar yields colonies up to 3mm in 24 hours - no growth on McConkey), confirmed with biochemical tests.

Differential diagnosis: HPAI, Newcastle disease, Colibacillosis, Fowl typhoid and Fowl pox.

Treatment

Doxycycline hyclate 200 mg, 250-600 g per 1000 liters of drinking water during 3-5 days (Equivalent to 20 mg per kg body weight), Or

Oxytetracycline HCl 200 mg, 80mg per kg body weight daily, during 3-5 days, or 40g per 100-150 liter of drinking water for 3-5 days. Or

Amoxicillin (as trihydrate) 150 mg, 16mg/kg BW or 100g per 100 liter of drinking water for 3-5 days. Or

Enrofloxacin 10mg/kg in drinking water for 3-5 days. Or

Chlortetracycline 10%, 20-60 mg/kg, daily in feed for 5 to 7 days or should be continued until sign disease are no longer apparent. Or

Flumequine 10 mg per kg body weight, 250 ml per 1000 liter of drinking water during 3-5 days.

Prevention and Control

Vaccination of pullet with live oral vaccine at 6 weeks, biosecurity, good rodent control, hygiene, predator control and proper disposal of dead bird is important as preventive measure.

Fowl Typhoid

Fowl typhoid is an acute or chronic septicaemic disease of mature chickens, potentially world-wide in distribution and frequently encountered in subsistence or semi-commercial flocks. The disease is responsible for serious economic losses in commercial units due to mortality (in both mature and immature flocks), loss of egg production and costs for prevention and treatment.

Etiology

The causative agent is *Salmonella gallinarum*: non-motile, gram negative, adapted to poultry.

Transmission

Vertical transmission occurs by the transovarial route, horizontal transmission takes place by direct contact between clinically affected and recovered carriers and by indirect contact with contaminated equipment, housing, litter, and clothing of personnel. The pathogen can remain viable in soil for up to a year.

Clinical symptoms

Acute

First sign is an increase in mortality, followed by drop in feed consumption, common features: Depression, ruffled feathers and closure of eyes. Respiratory distress with rapid breathing characteristic sign: watery to mucoid yellow diarrhoea, If no death within 2 -3 days, it may progress to chronic form.

Chronic

Progressive loss of condition, intense anaemia, shrunken, pale combs and wattles, rapid spread through the flock and result in losses of 50% or more.

In young chicks: Weakness, Somnolence (sleep), Anorexia Poor growth, huddling, gasping and pasty vent and death in up to 90% of the cases.



Fig.91. Fowl typhoid
Pasty vent,
In young chicks Pasting of vent
with chalky white excreta



Fig.92. Fowl typhoid
Pale comb in commercial layer,
Bovans Brown.

Postmortem lesions

Acute

Carcass appear septicemic and jaundiced, subcutaneous blood vessels: hyperaemic and engorge skeletal muscles: congested and dark in colour. consistent finding: dark reddish or almost blackish swollen friable liver with a characteristic *copper bronze sheen* (glistening brightness on the surface). Spleen: enlarged; small intestine: catarrhal enteritis with viscous, slimy, bile-tinged materials. characteristic feature: dark brownish bone marrow.

Chronic

Emaciated and anaemic carcass, greyish white necrotic foci in myocardium, intestines, pancreas and liver. characteristic feature: pericarditis with yellow turbid fluid in the pericardial sac and fibrin attached to the surface of the heart.

Diagnosis

Tentative diagnosis: history, signs and lesions, confirmatory diagnosis: isolation and identification of organisms Brilliant green and McConkey agar may be adequate investigation.

Differential diagnosis: Tuberculosis, Aspergillosis, Histomoniasis, Marek's disease or Reticuloendotheliosis.

Treatment

Oxytetracycline HCl 200mg, 80mg per kg body weight daily, during 3-2 days or 40 g per 100-150 liter of drinking water during 3-5 days. Or

Sulphachloropyrazine 300mg, 1000g per 100 liter of drinking water during 5 days for chicks and pullets as well as for laying hens.

Prevention and Control

Vaccination is the best method to control *Salmonella gallinarum*. If introduced control should focus on eradication of the disease through isolation and destruction of contaminated flocks, proper disposal of carcasses and disinfection of fomites.

Pullorum Disease (Dhaylin)

Pullorum disease, also known as bacillary white diarrhea (BWD), It is an infectious egg transmitted disease of young birds often characterized by white diarrhea & high mortality, local names of the disease is ‘**Dhaylin**’ or ‘**Shubanka cad**’ potentially world-wide in distribution but in practice is confined to non-commercial flocks in many countries, chickens and turkeys are most susceptible, although other species of birds can become infected.

Etiology

The etiologic agent of Pullorum disease is *Salmonella pullorum*, gram negative, non-motile and rod.

Transmission

Vertical transmission occurs by the transovarial route (through the egg, from hen to chick) and Horizontal transmission takes place by direct contact between clinically affected and recovered carriers and by indirect contact with contaminated feed, equipment, housing, litter, and clothing of personnel.

Clinical symptoms

In young chicks: If acute case: death occurs quickly or within two to three days after hatching without any observable signs, general signs in chicks after hatching are: loss of vitality (energy) and appetite, huddling, ruffled (dirty) feathers and diarrheal feces adhering around the cloaca.

In adult chickens: Affected birds may show dullness, loss of appetite, fever and yellowish diarrhea, the wattles and combs may become cyanosed, the disease may recur subsequently with reduced virulence and mortality.

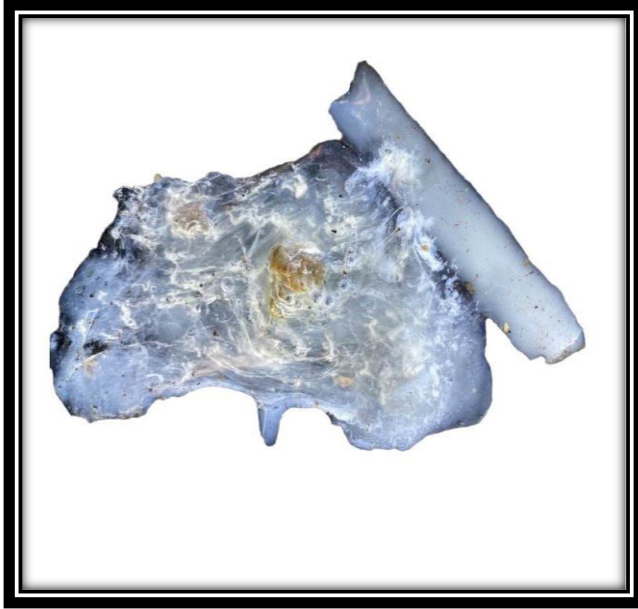


Fig.93. Pullorum disease
Chalk-white (urates) watery
mucoid diarrhoea.



Fig.94. Pullorum disease
Ruffled (dirty) feathers and
diarrheal feces adhering around
the cloaca.

Postmortem lesions

Chicks show enlargement of the spleen and liver, creamy or cheesy unabsorbed yolk in the abdomen and omphalitis is often present, chronic cases show abscessation of the viscera (heart, internal serosa, lungs, liver) and chronic caseous typhlitis characterized by grey casts in the caeca and enlargement and congestion of liver and greenish discoloration of the liver after exposure to air.

Diagnosis

Tentative diagnosis: by history, clinical signs and pathological lesions. Confirmatory diagnosis: by whole blood rapid agglutination test or the serum agglutination test and isolation and identification of the bacteria.

Differential diagnosis: Colibacillosis, Avian encephalomyelitis, Newcastle disease and Avian influenza.

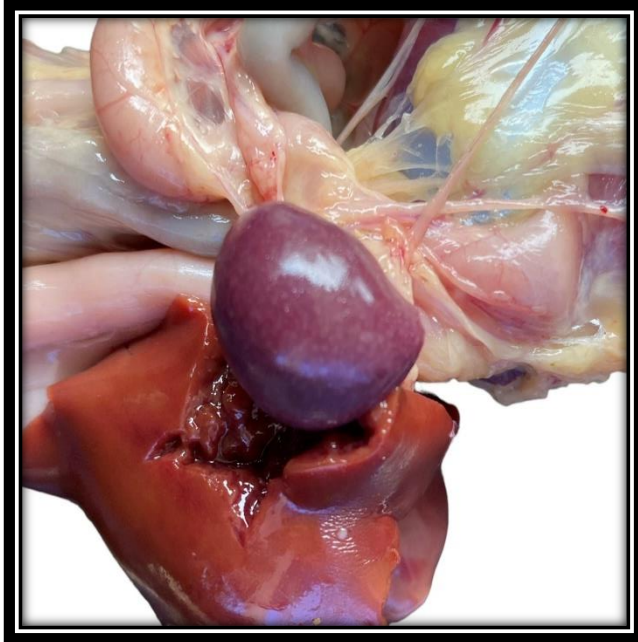


Fig.95. Pullorum disease
 Splenomegaly:
 Spleen is swollen, necrosed,
 mottled and brittle.



Fig.96. Pullorum disease
 Chick show enlargement of the
 liver in Koekoek.

Treatment

Trimethoprim-sulphaquinoxalene sodium (1.5), 30mg/kg PO in drinking water q 24 h, for 5 days or until two days after symptoms have subsided or 50-150 g per 200 liter of drinking water during 3-5 days. Or

Oxytetracycline HCl 500mg, 80mg per kg body weight daily, during 3-5 days, or 40g per 100-150 liter of drinking water during 3-5 days. Or

Gentamicin 5-10 mg per kg body weight in drinking water for 4-5 days.

Prevention and Control

Improvement of management, elimination of carriers, use of eggs from disease free flocks for hatcheries, chicks and poultries from disease free flocks, maintenance of sanitary environment., maintenance of a sound biosecurity program and proper disposal of dead birds.

Necrotic Enteritis

Necrotic enteritis (NE) is an acute enterotoxemia and the most common and financially devastating infectious disease in commercial layer pullets. Age affected: 3-6 weeks. The disease is often established after an outbreak of coccidiosis.

Etiology

Necrotic enteritis is caused by *C. perfringens* which is a gram-positive, rod-shaped anaerobe that forms oval sub terminal spores. It differs from most other clostridia in that the relatively large rods are encapsulated and non-motile.

Predisposing factors

Age and Immune Status: Young birds and those with compromised immune systems are more vulnerable to necrotic enteritis, as their natural defenses may not be fully developed or functional.

Stress: Environmental stresses, overcrowding, sudden changes in management practices, or other stressors can weaken the birds' immune system, making them more susceptible to necrotic enteritis.

Coccidiosis: Parasitic infections such as coccidiosis can damage the intestinal lining, providing an entry point for *Clostridium perfringens* and increasing the severity of necrotic enteritis.

Transmission

Clostridium perfringens are abundant in soil contaminants and can cause persistent outbreaks in successive flocks, pathogenic strains of *C. perfringens* may be introduced onto farms by deficiencies in hygiene and biosecurity and ingestion of the vegetative form of the organisms invariably results in colonization of the intestinal tract.

Clinical symptoms

Necrotic enteritis is characterized by acute, dark-colored diarrhea resulting in wet litter and in laying hens decreased egg production, suddenly increasing flock mortality of up to 1% per day after the first clinical signs appear sometimes summing up to mortality rates of 50% The birds have depression, ruffled feathers, lethargy, and inappetence.



Fig.97. Necrotic enteritis

The bird is depressed, eyes are closed, neck is extended, and head drooping, Koekoek.



Fig.98. Necrotic enteritis

Dark-colored diarrhea.

Postmortem lesions

The mucosa of the intestine shows changes ranging from focal hyperemia (redness) to mucosal ulceration, the intestine will be friable and distended with gas and gross lesions caused by toxins. In extreme cases, NE is characterized by extensive pseudomembranous enteritis which resembles a coarse yellow coating and liver is usually swollen & congested and focal hepatic necrosis may be observed.



Fig.99. Necrotic enteritis
Multiple petechial haemorrhages
in different areas along the
small intestine.

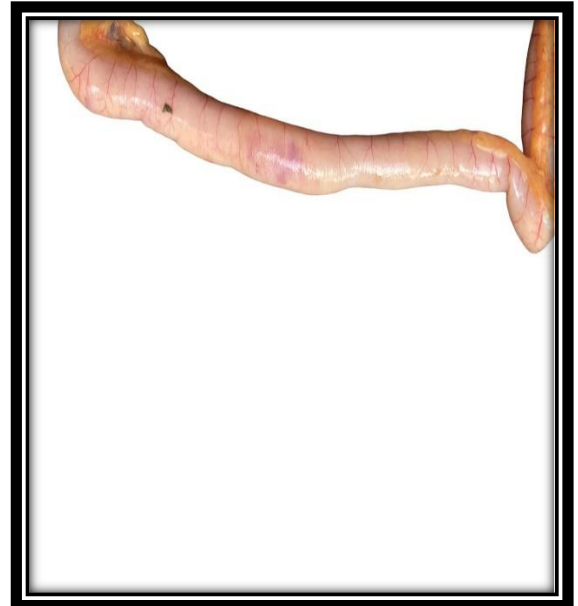


Fig.100. Necrotic enteritis
Focal hyperemia (redness) in
small intestinal.



Fig.101. Necrotic enteritis
Intestinal is filled with
brownish watery content,
mixed with gas bubbles.

Diagnosis

Presumptive diagnosis: clinical signs of dark-colored diarrhea, depression. Confirmation: based on gross lesions in small intestine and microscopic observation of gram-positive rods

Differential diagnosis: Coccidiosis and Ulcerative enteritis.

Treatment

Doxycycline hyclate 200 mg, 250-600 g per 1000 liters of drinking water during 3-5 days (Equivalent to 20 mg per kg body weight), Or

Amoxicillin 16mg/kg bw or 100g per 100 liters for 3-5 consecutive days. Or

Erythromycin, 500mg/gallon of water, for 7 days.

Prevention and Control

Prevention of coccidiosis, because *C. perfringens* is nearly ubiquitous, the key to controlling Necrotic enteritis is to prevent coccidiosis, which is the main predisposing factor for *C. perfringens* overgrowth and infection, minimizing stress factor, strict hygiene and disinfection measures, avoidance of animal by-products, rye, fish meal, wheat, barley in the diet and administration of probiotics or competitive cultures.

Staphylococcosis

Staphylococcosis is a chronic, debilitating disease affecting birds of all ages. Staphylococcal infections appear in five forms: septicemia, arthritis, omphalitis, bumblefoot and Gangrenous Dermatitis.

Staphylococcal septicemia: Infected birds typically have foul-smelling, watery diarrhea, many infected birds get swollen joints and/or experience a drop in egg production, because the bacteria is passed into the egg from infected hens, black rot might appear in eggs.

Staphylococcal arthritis: Follows the acute septicemia, birds become lame and develop breast blisters, infected birds are reluctant to walk, preferring to sit rather than stand.

Omphalitis: Infection of the yolk sac, is a common disease caused by *S aureus* in young chicks. navel infections can occur in young hatchlings via contamination of open navels and results in elevated mortality

Bumblefoot (pododermatitis): Is a localized staphylococcus infection of the foot. It is thought to be caused by puncture injuries, affected birds become lame from swollen foot pads.

Gangrenous Dermatitis: This occurs in birds of all ages, but is most common in broilers, and in growers between 15 to 20 weeks of age. The wing tips and the dorsal pelvic region are the sites affected. The affected sites are dark, moist, and gangrenous (decaying) in appearance

Etiology

The etiologic agent of Staphylococcosis is *Staphylococcus* contains dozens of species, but *S aureus* is the most pathogenic. *S aureus* is a gram-positive, catalase-positive, coccoid bacteria that appears in grape-like clusters on stained smears.

Transmission

Infection is usually by the respiratory route with an incubation period of 2-3 days seen after artificial infection, wounds, either accidental or induced by interventions such as beak trimming, toe trimming, parenteral vaccination, damaged skin due to nutritional deficiencies, may be a portal of entry with subsequent spread via the bloodstream to the typical sites of lesions.

Clinical symptoms

Increasing incidence of lameness occurs from 8 to 16 weeks and losses may attain 20% of the flock., affected birds are characterized by unilateral or bilateral hock arthritis and swelling. Occasionally there may be pododermatitis and ruffled feathers, affected birds invariably die of dehydration and persecution but rarely due to acute septicemia.



Fig.102. **Staphylococcosis**
Bumblefoot in a bovans brown.



Fig.103. **Staphylococcosis**
Lameness in a commercial layer,
Bovans Brown.

Postmortem lesions

Affected joints yield viscous yellow or green exudates or there may be abscess formation, lesions may include exudate on heart, liver, and yolk in chicks, puss in joints, breast blister, and foot pad in older birds (bumblefoot). Osteomyelitis (focal yellow areas of caseous exudate in the bones) and septicemia (congestion of liver, spleen, kidney and lungs) may occur.

Diagnosis

A presumptive diagnosis may be made on signs, history, lesions and isolation and identification of *Salmonella aureus*.

Differential diagnosis: Colibacillosis, *Salmonella* spp and *Mycoplasma synoviae*.

Treatment

Doxycycline hyclate 200 mg, 250-600 g per 1000 liters of drinking water during 3-5 days (Equivalent to 20 mg per kg body weight), Or

Oxytetracycline HCl 200 mg, 80mg per kg body weight daily, during 3-5 days, or 40g per 100-150 liter of drinking water for 3-5 days. Or

Amoxicillin 16mg/kg bw or 100g per 100 liters for 3-5 consecutive days. Or

Tilmicosin (as phosphate) 25%, 300ml per 1000 liter of drinking water during 3-5 days (20mg/kg body weight). Or

Erythromycin, 500mg/gallon of water, for 7 days. Or

Prevention and Control

Effective vaccination of immunosuppressive disease, biosecurity, good hygiene in the nest and the hatchery birds, proper sanitation and avoiding using sharp objects in houses or during transportation.

DISEASES CAUSED BY PROTOZOA

Coccidiosis

Coccidiosis is one of the most important protozoan disease of poultry worldwide. In Somali regional state of Ethiopia, it is a serious problem and one of the biggest causes of economic losses. Coccidiosis mostly occurs in rainy season. It is mostly a disease of young birds because immunity quickly develops after exposure and gives protection against later outbreaks.

Etiology

Coccidiosis caused by a group of protozoa of phylum *apicomplexa*, family *Emeridae*, Genus *Eimeria*. The parasite replicate within the epithelial cell characterized by intestinal damage, the poultry species are *host* and *site* specific, *Eimeria* spp that affect poultry *E. tenella*, *E. acervulina*, *E. necatrix*, *E. maxima*.

Factors contributing to outbreaks of clinical coccidiosis include: litter moisture content exceeding 30%, immune-suppression (Marek's disease, IBD, Mycotoxins), suboptimal inclusion of anticoccidials or poor mixing in feed and environmental and managerial stress.

Transmission

Coccidia are transmitted via the fecal-oral route, coccidial oocysts (eggs) are present in infected feces or the environment where feces is present (e.g. litter) and can be spread in blowing dust, boots, clothing, equipment. Birds ingest the eggs in feed, water, litter or soil and become infected. The eggs can survive up to four years in the environment.

Clinical symptoms

It is generally acute in onset and is characterized by depression, ruffled plumage, and diarrhea. Affected birds show decreased feed and water consumption and growth rate, followed by weight loss, decreased egg production, and increased mortality. Birds infected with *E. tenella* show pale color of comb & wattles, blood-stained fecal droppings and the area around the vent is stained with blood.



Fig.104. **Coccidiosis**
Yellow and foaming droppings,
early signs of Coccidiosis.



Fig.105. **Coccidiosis**
Ruffled plumage in a Bovans
Brown.



Fig.106. **Coccidiosis**
Blood containing faeces.

Table 1. **Three forms of Coccidiosis**

	Caecal coccidiosis	Intestinal coccidiosis	Rectal coccidiosis
Etiology	<i>E. tenella</i>	<i>E. necatrix</i>	<i>E. brunetti</i>
Symptoms	It is a severe disease associated with bloody droppings, high mortality, reduced weight gain, and emaciation.	It is associated with severe weight loss, morbidity and mortality. Intestinal coccidiosis occurs in relatively older birds. bloody droppings.	In moderate infections, the mucosa is pale bloody droppings.
Pathology	Caeca may be greatly enlarged and distended with clotted blood.	the middle portion of the small intestine is usually distended to twice its normal size (ballooning) and the lumen may be filled with blood.	Haemorrhages in a rectum, ceca and cloaca.

Postmortem lesions

Petechiae, thickening, ecchymoses, of caecal mucosa, accumulation of varying quantities of blood, caseous necrotic material in the caecum and haemorrhages in a rectum, ceca and cloaca.



Fig.107. Coccidiosis
Small intestine is greatly enlarged
and distended with blood.
Intestinal coccidiosis.



Fig.108. Coccidiosis
Haemorrhages in Small intestine
Intestinal coccidiosis.



Fig.109. Coccidiosis
Haemorrhages are patchial and
could be seen through the
intestinal wall.



Fig.110. Coccidiosis
large intestine filled with blood.
Rectal coccidiosis.



Fig.111. **Coccidiosis**
Clear Haemorrhages and
ballooning is typical of the
Intestinal coccidiosis



Fig.112. **Coccidiosis**
Haemorrhagic lesion in
lumen of small intestinal
(Close up view)



Fig.113. **Coccidiosis**
Caeca filled with blood.
Caecal coccidiosis.



Fig.114. **Coccidiosis**
Haemorrhages in a rectum,
ceca and cloaca.
Rectal coccidiosis.

Diagnosis

Presumptive diagnosis is based on clinical signs: presence of faeces with blood, dysentery diarrhoea suggests coccidiosis, In the laboratory a definite diagnosis by **Fecal flotation** and demonstration of oocysts in feces or intestinal scrapings.

Differential diagnosis: Ulcerative enteritis and Histomonosis.

Treatment

Sulfachlorpyrazine sodium monohydrate, 300mg/kg (0.03%) or 100 gm. Per 100 liter in drinking water for 3 days. Or

Trimethoprim-solphaquinoxaline sodium (1:5), 30 mg/kg PO in drinking water, Q 24 hr . for 5 days or until two days after symptoms have subsided or 50-150 g per 200 liter of drinking water during 3-5 days, but not exceeding 14 days altogether. adequate water should be given. Or

Amprolium HCl, 125mg/kg (0.0125%) in feed or 38.4 gm/ml in drinking water for 1-2 weeks or 1.2g/l/day for 3-5 days by continuous feeding. Or

Amprolium HCl, 200+Sulphaquinoxaline base 200 mg + Vitamin K₃ 2mg, 100g per 150 liter of drinking water for 5-7 days (for 2500 chickens of 5 week age or 1500 chickens of 10 weeks of age). Or

Sulphadimidine sodium 100mg/kg BW (1g powder per 1-2 liter of drinking water) in case of acute coccidiosis: administration during 3 consecutive days: after 2 days of no treatment, another 3day treatment. In case of subclinical coccidiosis: Administration for 3 consecutive days. Or

Toltraxuri 25 mg, 7mg per kg body weight or 100 ml per 100 liter of drinking water for continuous medication during 48 hours or 300 ml per 100 liter of drinking water for 8hours each day during 2 days. Or

Diclazuril 25 mg, 0.3 mg per kg body weight per day during 3 days. Or

Monensin, 20 mg/ kg. premix. Or

Cloplodol, 125 mg/ kg (0.0125%) of feed, by continuous feeding.

Prevention and Control

Management procedures which limit saturation of litter include: Appropriate installation and management of watering systems, nipple drinkers reduce spillage of water onto litter compared to bell and trough drinkers, acceptable ventilation rate, maintaining recommended stocking density. Inclusion of anticoccidials in diets at recommended levels, chemical & ionophoric anticoccidials for broilers in transport programs, synthetic coccidiostats for breeders and floor-reared commercial egg production flocks which allow the development of preimmunity.

Maintain good hygiene, good hygiene can greatly reduce the numbers of oocysts contaminating the environment. Moreover, good hygiene ensures that litter is kept dry so that it does not provide good sporulation condition.

Anticoccidial vaccines are appropriate for replacement breeding stock and roasters. This approach is cost-effective but requires experienced and monitoring especially if the vaccine is applied over feed. Intraocular administration by spray or the insertion of a gelatine cylinder impregnated with oocysts in the chick delivery box contributes to an even distribution of vaccine through the flock.

DISEASES CAUSED BY FUNGUS

Mycotoxicosis

Mycotoxicosis is a widespread problem in the poultry industry, is caused by ingestion of toxins produced by molds which contaminate cereals, some oilseeds, maize, wheat, rice, and peanut meal are most frequently implicated in cases of mycotoxicosis, Immature chickens and ducklings are most susceptible to mycotoxins, but age, current health and environmental stress also influence the response to various toxins.

Table 2. Different types mycotoxins

Toxin	Effect
Aflatoxin Produced by: <i>Aspergillus flavus</i> , <i>A. parasiticus</i> , and others	— Highly toxic, carcinogenic fungal metabolites — Primarily affects the liver, but immunologic, digestive, and hematopoietic functions may be involved <u>Clinical symptoms and lesions</u> — Unthriftiness to high morbidity and mortality, — Liver may be acutely reddened due to necrosis and congestion or yellow discoloration due to lipid accumulation; it may have hemorrhages of various sizes and patterns. — In chronic aflatoxicosis, the liver discolored yellow to gray and atrophied. — Tumor formation is rare
Fusariotoxigenesis genus <i>Fusarium</i> produces many mycotoxins	— Feed refusal, caustic injury (necrosis & ulceration) of the oral mucosa & areas of the skin in contact with the mold, acute digestive disease, — Mottling of the liver, atrophy of the spleen and other lymphoid organs, and visceral hemorrhages. — Diminished function of the bone marrow & immune system
Ochratoxin Produced By: <i>Penicillium</i> <i>viridicatum</i> , and <i>Aspergillus</i> <i>ochraceus</i>	— It is nephrotoxic, thus causes renal disease. — It can also affect the liver, immune system, & bone marrow. <u>Clinical symptoms and lesions:</u> — Reduced activity, huddling, hypothermia, diarrhea, & death are common. — Sublethal intoxication can impair weight gain, pigmentation, carcass yield, egg production, fertility & hatchability

Table 3. Different types mycotoxins

Toxin	Effect
Ergotism Produced by: <i>Claviceps</i> spp	— Affect the nervous system, & cause neurologic disorders — Cause Vasoconstriction and gangrene of the extremities — Influence neuroendocrine control of the anterior pituitary — The vasoconstrictive effect results in vesicles on the comb, wattles, face, and eyelids that rupture to form crusts, which may be followed by permanent atrophy and disfigurement. — Vesicles also develop on the legs & toes, and rupture to form ulcers. — Toes may be darkly discolored due to vasoconstriction & ischemia
Citrinin <i>Penicillium</i> spp. <i>Aspergillus</i> spp.	— It acts directly on the kidney and alter tubular transport processes. — Causes a diuresis that is manifested by watery fecal droppings and reductions in weight gain. — At necropsy, lesions involve chiefly the kidney.
Oosporein <i>Chaetomium</i> spp.	— Oosporein mycotoxicosis is characterized by visceral and articular gout related to impaired renal function and increased plasma concentrations of uric acid. — Water consumption increases and fecal droppings may be fluid.
Cyclopiazonic Acid <i>Aspergillus flavus</i>	— The proventriculus is dilated & the mucosa is thickened due to hyperplasia and ulceration. Lesions are also found in the gizzard, liver, and spleen.

Predisposing factors

The development of fungi is influenced by the, moisture content of the stored grain, temperature, condition of the grain going into storage, length of time the grain stored and amount of insect and mite activity in the grain.



Fig.115. Mycotoxicosis
Chicken die from acute aflatoxicosis
are usually found lying prone with their
feet stretched out behind them.
Courtesy of Dr. Muhiyadin Abdilahi

Diagnosis

History, signs, and lesions. Definitive diagnosis depends on a complete evaluation of necropsy, histopathology, bacterial and viral cultures, and serology accompanied by feed analysis from multiple samples.

Treatment

There is no specific treatment, concurrent diseases should be treated to alleviate additive or synergistic disease interactions, and substandard management practices corrected. In individual birds, give activated charcoal in the feed.

Control and Prevention

Detection of contaminated ingredients and exclusion from diets, to prevent proliferation of molds, store feed ingredients in less humid areas, poultry houses should have sufficient ventilation to avoid high relative humidity, Antifungal agents such as organic acids (propionic acid 0.5-1.5 g/kg) & Gentian violet can inhibit mold growth, inspect feed mills regularly and clean any feed residues and salvage of feed contaminated with aflatoxin is possible using high temperature ammoniation or adding commercial aluminosilicates to diets.

Candidiasis

Candidiasis is an opportunistic mycotic disease of the digestive tract of various avian species, including chickens, turkeys, and quail. Crop candidiasis is the most common candida-caused disease in poultry. These infections are called crop mycosis, thrush, moniliasis or sour crop.

Etiology

Candidiasis caused by the fungal yeast *Candida albicans* and usually occurs as a secondary infection.

Predisposing factors

Lack of good hygiene, prolonged antibiotic treatment, vitamin deficiency, severe parasitic infections and immuno-deficiencies. The production of a soluble endotoxin is also contributing to the pathogenicity of the agent. Candida species is an opportunistic pathogen and in a healthy bird they are found in small numbers. When bird's body suffers from a stressful condition, the immune system will be compromised. This condition will provide a good chance for candida species to grow and cause a clinical disease.

Transmission

Because of candida species are part of the normal flora, sources of infections are mainly endogenous, exogenous sources of can occur when a non-infected bird directly contact a diseased bird or indirectly by contacting the secretions and feces of a bird that is infected with candida species. Moreover, the organism can be transmitted through contaminated drinking water.

Clinical symptoms

Lack of appetite, weight loss, lethargy, stunting, depression and ruffled feathers, Enteric infection which is called crop mycosis, swollen crop, sour, disgusting -smelling breath, white patches in the mouth, thickening of the crop wall, dilation of the crop.



Fig.116. **Sour crop**
Sour, disgusting -smelling breath,
white patches in the mouth,
Bovans Brown.



Fig.117. **Sour crop**
White liquid exiting the beak.



Fig.118. **Sour crop**
Depression and swollen crop
in commercial layer, Bovans Brown.
(Lateral View).



Fig.119. **Sour crop**
Thickening of the crop wall,
dilation of the crop, Bovans Brown.

Postmortem lesions

Grossly at necropsy, lesions start as thickening of the mucosa due to hyphal growth. Later in the course of the disease, there are formation of yellowish to whitish pseudomembrane. The appearance of these lesions is often described as crud-like. Pseudomembranes are easy to peel and the mucus membrane under these diphtheritic materials usually eroded. These lesions are mainly seen in the crop but can be seen in the oropharynx and the proximal esophagus.

Diagnosis

Diagnosis of candidiasis can be confirmed by histologic demonstration of tissue invasion. microscopic lesions are characterized by epithelial hyperplasia, ballooning degeneration, and visualization of pseudohyphae and blastospores consistent with *Candida spp.*

Differential diagnosis: Wet pox, Vitamin A deficiency, and Infectious laryngotracheitis.

Treatment

Copper sulphate of 0.05% concentration in the drinking water is effective, antifungals in the feed or drinking water can be used such as: nystatin, parconazole, ketoconazole and gentian violet.

Prevention and Control

Good sanitary conditions in the flock environment along with elimination of any predisposing factors are enough for the prevention of clinical candidiasis. Infected birds must be removed from the rest of the flock as they are a potential source of infection. Fertile eggs should be dipped in an iodine solution to prevent transmission of the pathogen on the egg shell from the parent stock to newly hatched chicks

Aspergillosis

Aspergillosis also known as 'brooder pneumonia', Aspergillosis is an acute or chronic a fungal infectious disease, is primarily an infection of the respiratory system, of most bird species. Young chickens are very susceptible than old chickens, turkey, pheasants, quails, ducklings, and goslings may also become infected. The disease is world-wide in distribution but cases are more frequently diagnosed in tropical countries especially during warm and humid weather.

Etiology

Aspergillosis in poultry is caused by *Aspergillus fumigatus*, a fungal species under the genus *Aspergillus*.

Predisposing factors

Stress of cold, poor sanitary practices on the farm. high ammonia, and dusty environments, higher body temperature, chronic stress, overcrowding, malnutrition, vitamin deficiencies especially vitamin A and overuse of certain medications (corticosteroids) as well as respiratory irritants (disinfectant fumes and aerosol sprays).

Transmission

Transmission is by inhalation of fungus spores from contaminated litter (e.g. wood shavings, straw) or contaminated feed, horizontal transmission can occur in the hatchery or during handling, hatcheries may also contribute to infection of chicks.

Clinical symptoms

In chicks: Within the first 3-5 days newly hatched chicken infected in the hatchery show a very rapid and difficult breathing and start breathing with an open mouth (gasping) due to the gradual obstruction of the air passage.

Old chickens: Infected chickens are depressed, dull and stunted, laboured breathing, gasping, whistling rales, show sleepiness, lack of appetite, emaciation, increased thirst, develop eye swelling or blindness and show torticollis, lateral recumbency incoordination, tremors and paralysis, Other birds may remain subclinically affected for some times but later slowly develop breathing problems.



Fig.120. **Aspergillosis**
Breathing with an open mouth (gaspers),
local chick.



Fig.121. **Aspergillosis**
Laboured breathing, gasping,
Potchefstroom Koekoek.

Postmortem lesions

Numerous creamy colored nodules (yellow to grey nodules or plaques) in lungs, air sacs, trachea, plaques in peritoneal cavity, and occasionally in other organs including the brain and eye. Conjunctivitis/ keratitis, Brain lesions may be seen in some birds with nervous signs.



Fig.122. Aspergillosis
Lung shows the presence of cream color nodules.



Fig.123. Aspergillosis
Numerous firm, white to yellow fungal granulomas diffusely distributed on heart.



Fig.124. Aspergillosis
liver and gizzard show the presence of yellowish-white small nodules.

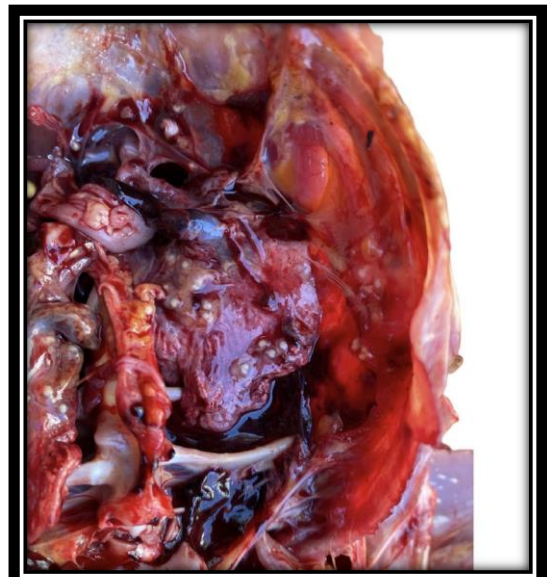


Fig.125. Aspergillosis
fungal granulomas.

Diagnosis

The presence of *Aspergillus fumigatus* can be identified microscopically or sometimes even with the naked eye in the air passages of the lungs, in the airsacs or in lesions of the abdominal cavity. Aspergillosis can be confirmed by culture using an appropriate fungal medium (Sabouraud's Dextrose Agar).

Differential diagnosis: Infectious bronchitis, Newcastle disease, Infectious laryngotracheitis and Nutritional encephalomalacia.

Treatment

No specific treatment available.

Prevention and Control

Removing the birds from contaminated area, removing infected birds from the flock, removing contaminated materials, increase ventilation and air exchange, good management, use only clean, uncracked eggs, mold-free litter and feed, keep water and feed systems clean, strict hygiene in breeder and hatchery management is necessary.

Dermatomycosis

Dermatophytosis, Favus, or ringworm, also known as white comb, is a chronic fungal disease of poultry. Dermatophytosis has a world-wide distribution. The disease occurs mostly in sporadic cases of backyard chickens; it has an important zoonotic potential to humans. This disease is characterized by the affection of chicken's skin, leading to loss of skin with scaly or crust lesions on the skin of the comb.

Etiology

Dermatomycosis is caused by the dermatophytic fungus *Trichophyton megninii*.

Transmission

The primary form of transmission is direct physical contact or indirect contact with other infected birds, animals or people; soil or fomites (inanimate objects such as feeder equipment, clothes). It spreads very slowly but the premises can become contaminated. Favus spreads gradually through a flock by direct contact, if left unchecked, and can produce ringworm lesions in their human handlers.

Clinical symptoms

Fungal infection that starts as small white spots on a chicken's comb and spreads to form large powdery white spots. White powdery spots on a chicken's comb and wattles, crusty and scabby comb and wattles, thick, crusty skin with hardened areas, chalky, white, moldy layers on the comb, feather loss and crusty skin around feather follicles, black spots on the comb from healing wounds, in severe cases it could attack the feather follicles and cause systemic disease.



Fig.126. Dermatophytosis
White powdery spots on a chicken's comb and wattle.

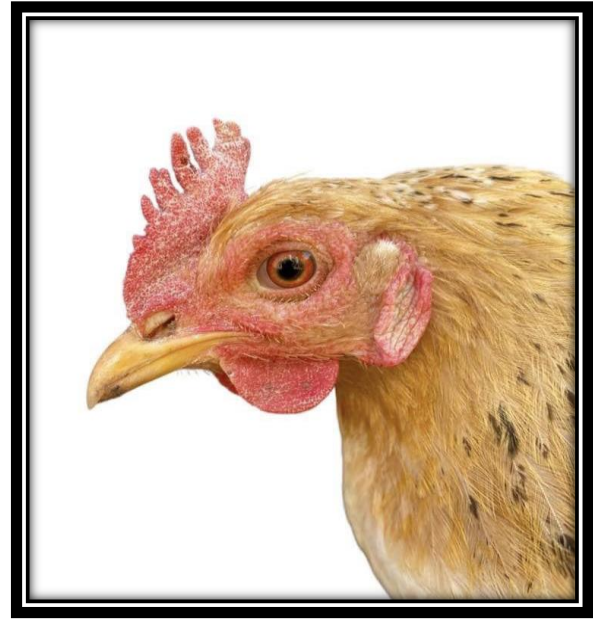


Fig.127. Dermatophytosis
Favus infection of the comb in a chicken.

Diagnosis

Skin scraping placed in a drop of 10% KOH and examined under the light of microscopy to identify the fungal spores.

Treatment

No treatment of good effect in this case but miconazole ointment has a good result to stop the disease.

Prevention and Control

Biosecurity precautions should be implemented to avoid introducing infected birds to the flock. When adding new birds to a flock they should be inspected as these may be carrying the fungus, Transport crates and other equipment should be thoroughly decontaminated and disinfected to prevent lateral transmission of the agent and Buildings should be thoroughly disinfected.

DISEASES CAUSED BY ENDOPARASITES

Gastrointestinal Parasitism

Poultry could be infected with nematode and cestode parasite. Among them: round worms, *heterakis gallinarum*, *syngamus trachea*, *Ascarids* and *capillarids* are economically important. *Raillietina spp* *Davainea proglottina* are important cestode species of poultry. The worm *syngamus trachea* inhabits the trachea and lungs of many domestic and various wild birds.

Clinical symptoms

Reduced appetite, reduced growth rate, diarrhea, anemia, gasping, coughing in case of *syngamus* infection), loss of condition and anorexia.



Fig.128. **Endoparasites**
Diarrhea and emaciation in
local rooster.



Fig.129. **Endoparasites**
Chronic Diarrhea is typical symptoms
of Gastrointestinal parasitism,
Bovans Brown.

Diagnosis

Based on clinical symptoms, post-mortem examination and identification of the parasite itself and eggs.

Differential diagnosis: Necrotic enteritis and Coccidiosis.

Treatment

Piperazine HCl 582-970 mg per kg body weight with drinking water or 195-390g per 100 liter of drinking water and deworm chicken at 4-6 week of age.

Plus

Phenothiazine (for cecal worms in chickens) 0.5g/bird individual treatment or in dry or wet mash feed. Once a month to control ascaris. Or

Phenothiazine 0.5gm/head or combined with piperazine (0.5-0.51% & 0.11% in drinking water) as a 1-day treatment, remove both heterakid and ascarids. Or

Ivermectin 10mg, 0.25-0.32 mg per kg body weight. C/I don't use in breeders in productions or layers producing eggs for human consumption. Or

Coumaphos, 0.004% in feed for 10-14 days for replacements, or 0.003% in feed for 14 days for layers, is used against capillarids.

Prevention and Control

Strict sanitation and disinfection measures of bedding as well as feeders and drinkers, control of secondary hosts such as beetles roaches, grasshoppers and earth worms, use of a deworming program, and avoidance of multi age farming.

Table 4. Endoparasites

Endoparasites	Pathology
Ascaridiasis	Etiology — Ascaridia galli Transmission — Fecal oral route, ingestion egg of ascaridia gali Clinical symptoms — Light to medium infestations may not produce clinical signs; however, heavy infestations may cause diarrhea, intestinal occlusion, intussusceptions, emaciation, anemia, and death. There is reduced egg production, and birds appear unthrifty. Postmortem lesions — Lesions caused by this worm are cattarhal or hemorrhagic enteritis. This parasite has been implicated in some cases of egg yolk peritonitis. Diagnosis — Diagnosis is through finding eggs in feces, or worms during postmortem examination. — Differential diagnosis: Hartertia gallinarum. Treatment — Piperazines Prevention and Control — Separate young birds from old birds when kept in enclosures. Moisture levels and ventilation should be monitored.
Capillariasis	— These are small, hair-like worms found in the digestive tract. Etiology — Capillaria species Transmission — Fecal oral route. Clinical symptoms — The birds appear weak and emaciated. Capillaria contorta and C. annulata cause cattarrhal inflammation, and thickening of the crop and esophagus. C. caudinflata, C. bursata, C. obsignata and C. anatis are associated with hemorrhagic enteritis and bloody diarrhea Diagnosis — Diagnosis is through finding eggs in feces, fecal flotation or worms during postmortem examination. Treatment — Treatment is with coumaphos and febendazoles Prevention and Control — Control and prevention is by separation of birds from possible transport and intermediate hosts, and effective cleaning of poultry houses and premises.

DISEASES CAUSED BY ECTOPARASITES

Scaly Leg Mite Infestation (Qaranjo)

The scaly leg mite, also known as knemidocoptiasis, cutaneous knemidokoptosis are a relatively common ectoparasite of chickens, affecting backyard and free-range flocks in the Somali region state of Ethiopia. *K. mutans* are sarcoptic mites (burrowing mites) which burrow into the cornified epidermis of the skin, causing tissue damage. The mites are most often found on the legs and feet, but occasionally the comb, wattle, neck and/or beak.

Etiology

Knemidocoptes mutans, is a small, spherical, sarcoptic mite that usually tunnels into the tissue under the scales of the legs. It is rare in modern poultry facilities but not uncommon in backyard flocks.

Transmission

Knemidocoptes mutans are spread between chickens by direct contact with infected birds, they are initially introduced into a flock through adding a new chicken who is infected already, or exposure to wild birds or rodents.

Clinical symptoms

Heavy infestation will cause raised scales, crusting and distortion of the skin, prolonged infestations with *K. mutans* can lead to necrosis of the toes, lameness, and permanent deformation of the feet. In addition, the compromised skin barrier puts the birds at higher risk of developing secondary bacterial infections, Birds stop feeding, and death can result after several months. This mite may occasionally attack the comb and wattles.



Fig.130. Scaly leg mite infestation
Rooster is normal while hen is infected
Scaly leg mite.



Fig.131. Scaly leg mite infestation
Scaly leg mite in hybrid rooster.



Fig.132. Scaly leg mite infestation
A severe case Scaly leg mites

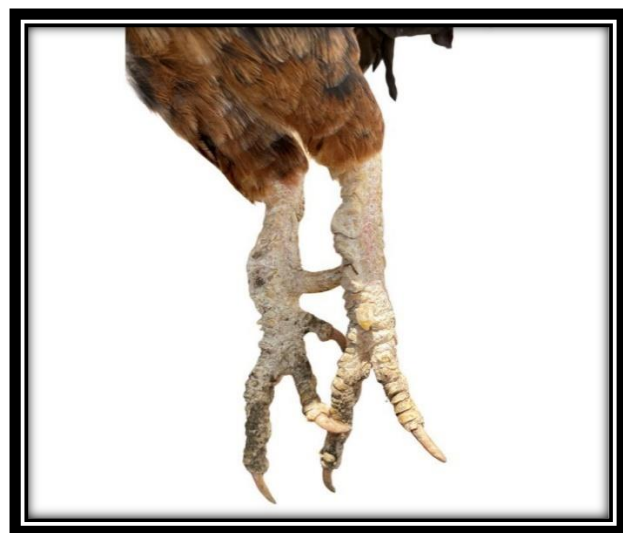


Fig.133. Scaly leg mite infestation
A few scales raised, then the mites
spread up the leg and down the toes.

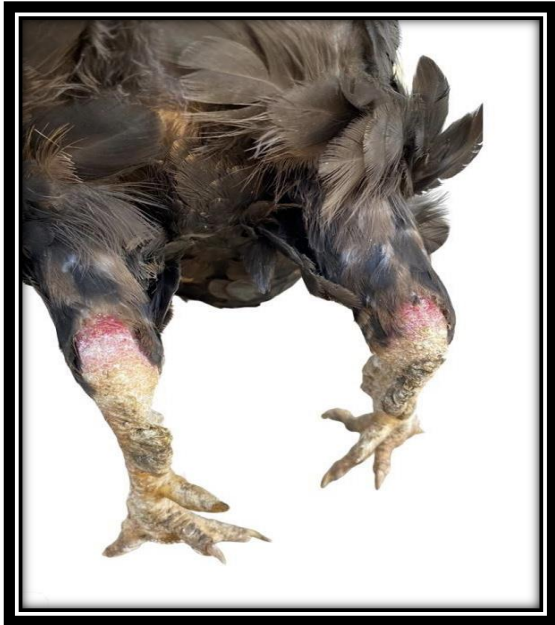


Fig.134. Scaly leg mite infestation
Raised scales, crusting and distortion of the skin.



Fig.135. Scaly leg mite infestation
Necrosis of the toes.



Fig.136. Scaly leg mite infestation
Severe case of scaly legs, cause a claw to drop off.



Fig.137. Scaly leg mite infestation
Permanent deformation of the feet.

Diagnosis

The gross appearance of the lesion is pathognomonic. The diagnosis may be confirmed by direct microscopy, is considered to be a standard and reliable diagnostic technique for rapid diagnosis in which skin over the burrow of a mite, identified by its appearance, is scraped or the contents removed on a sterile needle and the material sent to a laboratory.

The sample is placed on a slide and 5 drops of 10% potassium hydroxide or normal saline are added. The sample is covered with a cover slip and visualized under a microscope for the presence of mites, larvae or ova. *Knemidocoptes mutans* characterized by short stubby legs, the terminal anus, and the dorsal surface covered by faint striation. Mid-dorsally, the pattern of dorsal striations is broken in a plate or scale-like pattern. The body has no scales or spines.

Treatment

Treatment involves a combination of softening the scales, via gentle regular washing, together with application of treatment to suffocate or kill the mites, such as Vaseline, paraffin, mineral oil, or an insecticide. Individual birds should be treated with oral or topical ivermectin or moxidectin (0.2 mg/kg), 10% sulphur solution, or 0.5% sodium fluoride.

Prevention and Control

Provide your flock with a dust bathing area, maintain a clean environment, keep rodents out of coops, Scaly leg mite infestation in chickens, prevent contact with wild birds, thoroughly clean and disinfect any tree branches brought in to be used as perches for birds and reduce stress. For control, affected birds should be culled or isolated, and houses should be cleaned and sprayed frequently.

Echidnophaga Gallinacea (Qirinqiir)

Echidnophaga gallinacea, also known as the hen flea or sticktight flea, is commonly found in lowland areas with warmer climates. Common problems in Somali backyard chickens in different zones of Somali regional state of Ethiopia. *Echidnophaga gallinacea* infestations are known as "**Qirinqiir**" among Somali people. Stick tight fleas are small brown-black parasites visible in clusters firmly attached to the skin, typically around the eyes, on the face, comb and wattles. Adult fleas feed on blood and stay firmly attached to the bird, while eggs and nymphs remain in the surrounding environment.

Etiology

Etiological characteristics: Adults are approximately 1.5 to 4 mm in length, laterally flattened, and are black to brown in color, their head is sharply angled at the front, there are no genal or pronotal ctenidia. On the head behind the antennae are two setae, and in females, a well-developed occipital lobe. Thoracic segments are narrowed dorsally. Mouthparts appear large, extending the length of the forecoxae and projecting from the head conspicuously. The maxillary laciniae are broad and coarse.

Transmission

Echidnophaga gallinacea can be transmitted between birds through direct contact or by sharing living spaces. They can also be spread indirectly through contaminated bedding, nesting materials, or other surfaces that have come into contact with infested birds.

Clinical symptoms

The adult fleas attach to the skin around the face and head, causing severe irritation, restlessness, anaemia, nodular formation. In severe infestation of the fleas around the eyes cause ulcerations, blindness and They can cause blood loss, anemia, and even death due to starvation. The skin over the nodules often becomes ulcerated, and young birds may be killed by heavy infestations.



Fig.138. **E. Gallinacea infestation**
In severe infestation of the fleas
Feeds on blood, cause blood loss,
anemia, pale comb & wattle.



Fig.139. **E. Gallinacea infestation**
Fleas around Nostril in local chick.



Fig.140. **E. Gallinacea infestation**
Fleas embedded around the eyes,
comb and wattle in Somali chicken.



Fig.141. **E. Gallinacea infestation**
Clusters of fleas attached to the
featherless skin around the eyes.
in Shiinawi, Somali chicken.



Fig.142. **E. Gallinacea infestation**
Fleas embedded around the eyes and
Wattle in commercial layer,
Bovans Brown.



Fig.143. **E. Gallinacea infestation**
Fleas around the eyes in Koekoek.



Fig.144. **E. Gallinacea infestation**
Clusters of fleas attached to the
featherless skin around the eyes.
in commercial layer, Bovans Brown.

Diagnosis

This is based on history, clinical signs, and identification of fleas or flea feces on birds.

Treatment

Removal of the adult fleas from the chicken using tweezers, followed by applying a topical antibiotic ointment to help minimize risk of secondary infection.

Prevention and Control

Managing fleas on birds requires an integrated approach. Both the host bird and the environment must be treated at the same time to be effective. Birds and surroundings should be sprayed with insecticide. Control the fleas by treating the birds, removing them from the infested area for three weeks, and treating the area (removing fecal droppings and litter, and spraying a suitable insecticide) and birds (again) before returning them.

Domestic animals having contact with poultry should be treated at the same time. A range of insecticides can be used for flea control on the birds: organophosphates, carbamates, pyrethrins, and pyrethroids.

The most important flea control measures are to remove infested litter and to dust the litter surface with carbaryl, coumaphos, malathion, or pyrethroids to kill immature fleas. Insect growth regulators such as methoprene are also effective. Sticktight fleas can be controlled by topical application of insecticides.

Table 5. Ectoparasites

Ectoparasites	Pathology
Chicken Mite	— Dermanyssus gallinae (Red mite) infests chickens, turkeys, other birds in breeder and small farm flocks, but rare in modern commercial cage-layer operations. Chicken mites are nocturnal feeders that hide during the day under manure, on roosts, and in cracks and crevices of the chicken house, where they deposit eggs. They are the most significant economically. Treatment and Prevention — Accaricides for ticks can be used and miticide spray treatments must be applied with sufficient force to penetrate the feathers in the vent area. see below: — Dichlorovos 23 EC, diluted 5oz/gallon, 1 gallon/100 birds or 1-2gallon/1000 sq. ft of litter. • Tetrachlorinfos 50 WP, 6.5 gallon, 1 gallon/100 birds or 1-2gallon/1000 sq. ft of litter
Common Chigger	— Trombicula alfreddugesi and other chigger species (harvest mites, red bugs) infest birds as well as man and other mammals. Heavily parasitized birds become droopy, refuse to eat, and may die from starvation and exhaustion. Larvae may be found either singly or in clusters on the ventral portion of the birds. Treatment and control — Use miticide sprays like chicken mite above. Control on the range is aided by keeping the grass cut short and dusting with sulfur or Malathion.
Lice	— Heavy infestation of poultry and other avian species with Mallophagia lice. Infested poultry results in reduced egg production and weight loss. The skin irritations are also sites for secondary bacterial infections. Diagnosis — The eggs or adult lice could be observed on the skin or feathers. Treatment and Prevention — After houses are cleaned, poultry house should be treated thoroughly (using a high-pressure sprayer) with acaricides like: Coumaphos 20% E.C., is used in concentrations of 250 ppm, to be sprayed on walls, ceilings, cracks, and crevices.

Dust bathing

One way to control external parasite is to ensure that a flock has access to dry dust or fine sand for bathing.



Fig.145. Ectoparasites

Chickens bathe by flapping their wings and kicking their legs to cover themselves with dust. The preening that follows helps control external parasites, Bovans Brown.

NUTRITIONAL DISEASES

Rickets

Rickets is a condition in which there is a failure or inadequate deposition of minerals in the bones, that is, calcium and phosphorus, birds under four weeks of age are most susceptible, but the condition may occur at any age.

Etiology

The etiological factors include dietary Ca, P and vitamin D3 deficiency; disturbed Ca/P ratio, or impaired absorption of one of these elements.

Clinical symptoms

General weakness, poor growth, skeletal deformities, lameness, swollen Joints, soft beak, poor feather quality and delayed egg production.

Postmortem lesions

The bones do not break with a sudden sharp sound, because they become soft and rubbery from poor mineralization, the beak and shanks are soft and pliable. In birds with severe rickets, the keel bones are not straight, but twisted, bent, or curved, ribs are 'beaded'. that is, heads of the ribs are enlarged as nodules the legs are bent, and ends of the long bones enlarged.

Diagnosis

A tentative diagnosis is based on clinical signs and visible signs of bone abnormalities.

Treatment

Administer Vitamin 03 in the recommended dose 2-3 times through drinking water. Replace the suspected feed with the known well balanced and properly mixed feed.

Prevention and Control

Ensure adequate supply of Vitamin 03 and Calcium and Phosphorus in 2: 1 ratio in the feed. Mineral premixes should be properly mixed for even distribution.



Fig.146. Rickets

The birds are unable to stand, toes are curled inward, Somal local chicken.



Fig.147. Rickets

Suffering from severe calcium deficiency, the birds are unable to stand, toes are curled inward. Potchefstroom Koekoek.

Table 6. Nutritional Diseases

Nutritional Diseases	Pathology
Vitamin A deficiency	— vitamin A is essential in poultry diets for growth, good vision, and integrity of mucous membranes. vitamin A is the most challenging, because it is most likely to be deficient in poultry. Etiology — Low levels of vitamin A in the feed. Clinical symptoms — In chicks, symptoms include loss of appetite, poor growth, ruffled feathers, sleepiness, and weakness. Deficient chicks are very susceptible to infections, due to inadequate antibody production. — In adult chickens, changes of vitamin A deficiency first appear in the pharynx and oesophagus. Egg production decreases sharply and hatchability is reduced. Postmortem lesions — Inner lining of the oesophagus shows tiny white raised granules. Diagnosis — This is based on the characteristic postmortem findings in the oesophagus. Treatment — Administer a stabilized vitamin A preparation at a level of about 10,000 IU vitamin A per kg of feed Prevention and Control — Use stabilized vitamin A preparation in the feed. Add adequate levels of antioxidants in the feed. Provide adequate storage for feed and feed ingredients.
	Clinical signs — Young chicks, as early as 1-week-old, exhibit curling of the toes, inability to walk and sometimes diarrhea. Treatment and control — Administering vitamin B preparations brings a rapid cure. Only in advanced cases will birds be dehydrated and emaciated, requiring further treatment. It is important to ensure adequate vitamin B levels not only in starter and grower diets, but also in the diet of parent breeders.

Table 7. Nutritional Diseases

Nutritional Diseases	Pathology
Vitamin D3 deficiency Rickets, “Rubber Legs”	Clinical signs and gross lesions — Young chickens, 2 to 5 weeks of age, with vitamin D3 deficiency are unable to stand and have very soft, pliable, legs and beaks. The rib joints are swollen like beads and curved inward, the breastbone often twisted. In layer chickens, vitamin D3 deficiency causes soft-shelled eggs and a drop in production. Treatment and control — Vitamin D3 can be given as treatment, usually in combination with calcium and phosphorus.
Vitamin E deficiency Crazy Chick Disease, Encephalomalacia	Clinical signs and gross lesions — Vitamin E deficiency in chickens affects the brain, causing degeneration, edema and hemorrhage, especially in the small brain (cerebellum). Affected young chicks appear unable to walk, they fall on their sides or stand with their heads between their legs. The cerebellum shows gross swelling, with yellow or brown discoloration and pinpoint hemorrhages may be observed. Encephalomalacia can also be found in mature chickens. Treatment and control — Adequate levels of vitamin E and selenium in the diet of chickens and their parent breeders is of prime importance. Treatment of affected birds with vitamin E preparations (alpha-tocopherol) is effective if the condition is not too advanced.

MISCELLANEOUS CONDITIONS

Cannibalism

Cannibalism is the act of one individual of a poultry species consuming all or part of another individual of the same species as food. In the Somali Regional State of Ethiopia, Cannibalism in poultry is a common problem in dense poultry populations, such as found in commercial settings, but occurs only sporadically in village flocks.

Etiology

Overcrowding, excessive light, and nutritional imbalances are directly correlated with its occurrence. Other factors that predispose to Cannibalism are overly fat pullets where mucosa protrudes from the vent during and after egg laying, insufficient feeder space, mineral and vitamin deficiencies, skin injuries, and failure to remove dead birds daily.

Clinical symptoms

Severely damaged or missing feathers, and hemorrhages or scabbing on the head, wings, tail, vent, and other body areas are characteristic signs, birds dying of Cannibalism may have anemia, blood stained feathers, and affected hens decrease or end their egg production.

Feather Picking is a behavior expressed by dominant birds toward subordinates. It varies from pecking to plucking the feathers of subordinate birds. Feather picking, if severe, can result in fatal hemorrhage in any age of bird.

Vent picking occurs in laying birds. It is common when birds raised in floor systems lay their eggs on the floor in crowded areas. This occurs after oviposition, when exposed mucus membranes stimulate pecking by other birds. Vent pecking may result in birds eating off portions of the intestines, resulting in death of the affected bird.

Toe picking occurs commonly in young birds, while head picking is more common in older birds, especially those housed in cages.

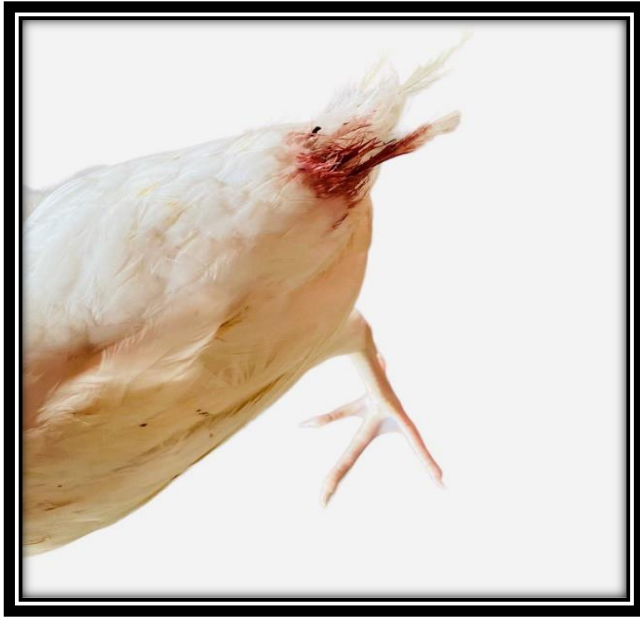


Fig.148. **Cannibalism**
Feather Picking in Dekalb White.



Fig.149. **Cannibalism**
Vent picking in Bovans Brown.



Fig.150. **Cannibalism**
Vent picking, resulting in death of the
affected bird, Bovans Brown.



Fig.151. **Cannibalism**
Vent gleet is factor that predispose to
Vent picking.

Diagnosis

Diagnosis is based on the appearance of typical injuries

Treatment

Wounds on individual birds can be treated with wound management and spraying antibiotics.

Prevention and Control

Effective methods to prevent and manage cannibalism in poultry:

Provide Adequate Space: Overcrowding is a common trigger for Cannibalism in poultry. Ensure that the birds have enough space to move around comfortably in the coop or housing area.

Ensure Proper Nutrition: A balanced and nutritious diet is crucial for the overall health of poultry. Make sure the birds have access to quality feed that meets their nutritional requirements.

Manage Stress: Minimize stress factors in the flock, such as sudden changes in environment, loud noises, or predator threats. Ensure a calm and peaceful environment for the birds.

Trim Beaks: Trim the tip of the beak at one-day old and repeat between 6 and 12 weeks age. Cautery often is required to provide hemostasis. Beak trimming can help reduce the severity of Cannibalism by minimizing the damage caused by aggressive pecking.

Separate Aggressive Birds: If certain birds are exhibiting aggressive behavior and causing harm to others, separate them from the rest of the flock to prevent further incidents of Cannibalism.

Monitor and Remove Injured Birds: Regularly inspect the flock for any injured or weak birds. Promptly remove such birds from the flock to prevent them from becoming targets of cannibalistic behavior.



Fig.152. Cannibalism
Treatment of wound caused by
Feather Picking in Dekalb White.



Fig.153. Cannibalism
Beak trimming in pullet of
Bovans Brown to prevent cannibalism.

.

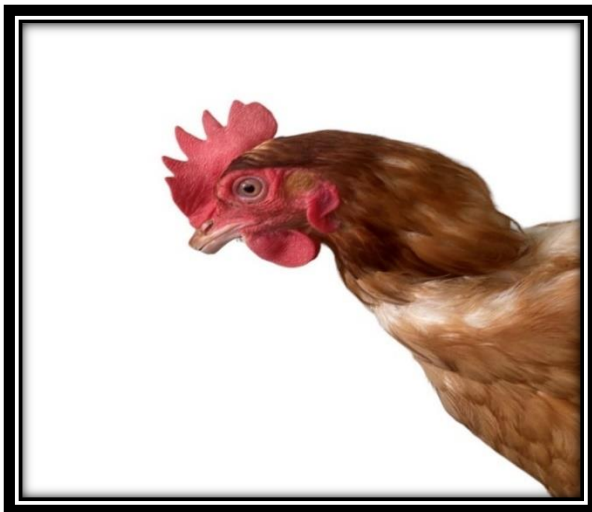


Fig.154. Cannibalism
Beak trimming when chicken start
laying eggs, Bovans Brown.

.

Vent Gleet

Vent gleet also known as cloacitis is essentially an inflammation of the cloaca, it involving the digestive and reproductive systems. poultry of all ages can be susceptible, but it is not contagious

Etiology

One of the most prevalent causes of vent gleet is fungal and yeast infections. Most typically, it came from the Candida species. Ingesting moldy or spoiled food-especially corn, also contaminated water and Poor sanitation can lead to vent gleet in chickens due to overcrowded and unhygienic conditions.

Clinical symptoms

Symptoms typically include discolored discharges from the vent accompanied by redness and swelling of this area, white discharge from the vent, missing or soiled feathers around the vent, red or swollen vent- can be bloody if severe case, loose stools, decrease or cessation of egg laying, depression, decreased appetite or increased appetite, loss of weight, whitish patches, pasting of vent feathers and swollen bloated abdomen.

Treatment

Bathe the chicken to help cleanse and soothe the affected area. Nystatin liquid suspension orally is very effective for 7-10 days.

Prevention and Control

Add probiotics to their diet by enriching their food or feeding them plain unsweetened yogurt with live and active cultures once per week. This helps to restore the balance of the normal flora. Clean the wateres regularly with distilled vinegar. keep the coop and run clean, practice good hygiene. Keep the coop and run clean and dry, never feed the chickens kitchen scraps you would not eat yourself, keep chicken feed dry and stored in weather tight metal garbage cans. and discard any questionable or moldy feed.



Fig.155. Vent gleet
White discharge from the vent,
in a Bovans Brown.



Fig.156. Vent gleet
Vent accompanied by redness and
swelling in a commercial layer,
Bovans Brown.



Fig.157. Vent gleet
Redness and white discharge
from the vent in a Bovans Brown.



Fig.158. Vent gleet
Soiled feathers around the vent
in a commercial layer, Bovans Brown.

Prolapsed Vent

Prolapsed vent, also known as a cloacal prolapse, can occur in chickens and is a common condition in laying hens. Prolapse vent in chickens is an avian anomaly wherein a hen's vent or cloaca is inverted and pushed to the outside of the body. Often characterized by a collection of red tissue hanging out of the hen's posterior.

Etiology

Straining, obesity, dietary Issues: poor diet, low calcium levels and nutritional imbalances, Infections, high light intensity and excessive egg production.

Clinical symptoms

The first signs of a prolapse are blood-streaked eggs, lack of appetite, unwillingness to eat, self-isolation, unwillingness to socialize, lethargy and lack of energy, huddling while puffing out feathers, the most obvious symptom is the protrusion of the cloaca (vent) from the bird's body, the prolapsed tissue may appear red, swollen, and possibly bloody. Hens may have difficulty laying eggs or may stop laying.

Treatment

Isolation: Isolate the affected bird in a quiet and clean area to minimize stress.

Gentle Cleaning: Clean the prolapsed tissue with warm water and gently push it back into place. Apply a lubricant if needed.

Prevention and Control

Providing a comfortable nesting environment, maintaining proper weight, and monitoring egg production and dietary changes: ensure the bird is receiving a balanced diet with adequate calcium levels to support muscle function.

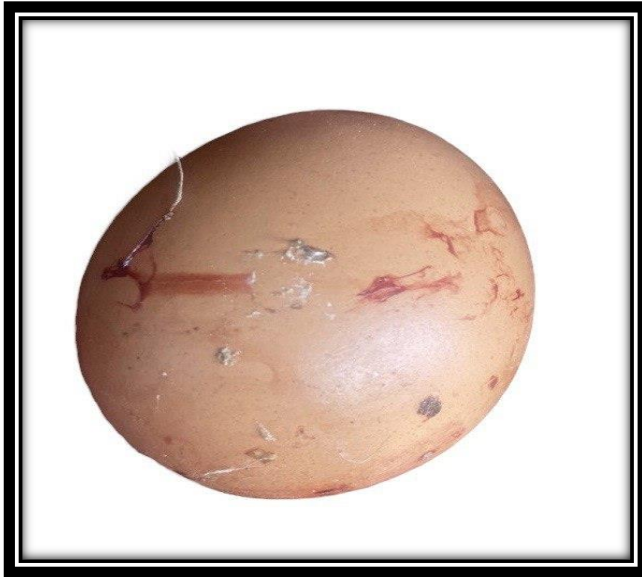


Fig.159. Prolapsed vent

The first signs of a prolapse are blood-streaked eggs.



Fig.160. Prolapsed vent

The prolapsed tissue may appear red, swollen, and bloody.



Fig.161. Prolapsed vent

Cloaca is inverted and pushed to the outside of the body in a Bovans Brown.

Courtesy of Dr. Abdirahman D. Ibrahim



Fig.162. Prolapsed vent

After treatment in a commercial layer, Bovans Brown.

Courtesy of Dr. Abdirahman D. Ibrahim

Feather Loss

Feather loss in chickens can be caused by various factors, common causes of feather loss in chickens:

Molting: Molting is a natural process in which chickens shed old feathers and grow new ones. During molting, natural Moulting after 60 weeks of age

Parasites: External parasites, such as mites and lice, can cause feather loss in poultry. These parasites irritate the birds' skin, leading to excessive preening, pecking, and scratching, which can result in feather damage and loss.

Cannibalism: Feather pecking and cannibalism can occur in poultry flocks, resulting in feather loss among affected birds, aggressive behavior, stress, overcrowding, or nutritional deficiencies can contribute to cannibalistic behaviors.

Skin conditions: Skin infections, fungal infections, or dermatitis can cause itching and discomfort in poultry, leading to excessive grooming and feather loss. Bacterial infections, such as feather follicle infections, can also result in feather loss.

Nutritional deficiencies: Inadequate nutrition, such as protein, vitamins, or minerals deficiencies, can affect the health of feathers and lead to poor feather quality and loss. Ensuring a balanced diet is essential for feather health.

Environmental factors: Extreme temperatures, poor ventilation, high humidity, or dirty bedding can contribute to stress in poultry, leading to abnormal feather loss.

It is important to identify the underlying cause and implement appropriate measures. This may include improving nutrition, regular monitoring, proper nutrition, parasite control, managing stress factors, and promoting overall flock health and welfare.



Fig.163. Feather loss
Feather loss on wings of Bovans Brown.



Fig.164. Feather loss
Feather loss in Somali backyard chicken.



Fig.165. Feather loss
Feather loss in local chick.



Fig.166. Feather loss
Feather loss in Bovans Brown.



Fig.167. Feather loss
Feather loss in commercial layer,
Bovans Brown.



Fig.168. Feather loss
Feather loss on neck of local rooster.

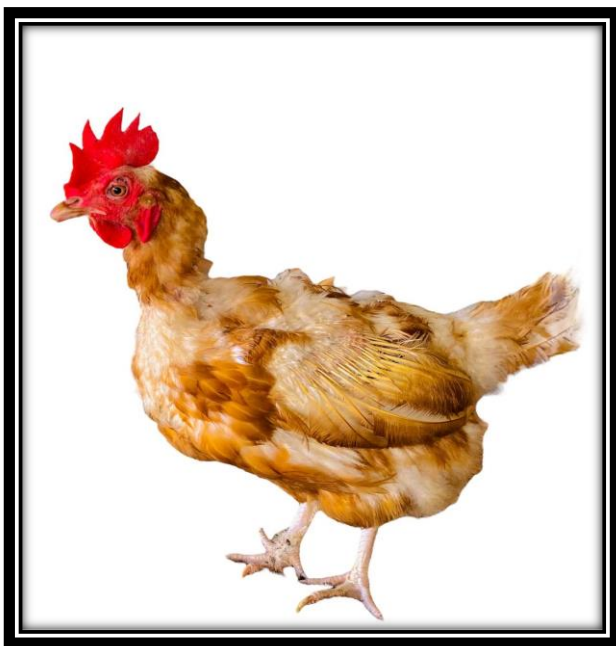


Fig.169. Feather loss
Feather loss in Bovans Brown.



Fig.170. Feather loss
Feather loss on back of hybrid
chicken.

Comb

Comb refers to the fleshy, often brightly colored, crest located on top of the chicken's head. Combs serve various functions in chickens, including

Regulation of Body Temperature: The comb acts as a thermoregulatory organ, helping chickens regulate their body temperature. In hot weather, blood flow to the comb increases, allowing heat to dissipate from the body. Conversely, in cold weather, blood flow decreases to conserve heat.

Signaling and Communication: The comb can be an important visual cue in signaling dominance, courtship, or social hierarchy within the flock. A vibrant, erect comb may signify good health and fertility, attracting potential mates.

Health Indicator: Changes in the color, texture, or appearance of the comb may indicate the overall health status of the bird. Pale, discolored, or droopy combs can be a sign of illness, dehydration, or nutritional deficiencies.

There are several different types of combs found in various chicken breeds, common types of combs found in Somali regions including:

1. **Single Comb:** The single comb is the most common type of comb and is found in many chicken breeds. It is a flat, single row of evenly serrated, upright points.
2. **Rose Comb:** The rose comb is compact and cushion-shaped, with a cluster of small, rounded protrusions. It is lower and broader than the single comb, giving it a neat and tidy appearance.
3. **Pea Comb:** The pea comb is characterized by three distinct, evenly spaced rows of small, round, and smooth peas. This type of comb is shorter and broader than the single comb..
4. **Strawberry Comb:** The strawberry comb is a variation of the rose comb, characterized by a rounded, bubbly appearance resembling a strawberry.
5. **Cushion combs:** are small, close to the chicken's head, and look like a cushion sitting on top of the head. Most combs are bumpy and textured, but the cushion comb is smooth

Types of combs



1. Single comb (Dhoor-taag)



2. Pea comb (Dhoor-jiif)



3. Cushion comb (Rudcane)



4. Strawberry comb (Jarane)



5. Rose comb (Dhoor-waran)



6. No comb (Guudlay)

Comb health indicator



Rosy red comb: Desired colour, Indicator of a Healthy chicken.



Pale pink: sign of anemia often caused by Internal or external parasite. Other reasons: Non-laying pullet, molting season, heat stress, diseases or having difficulties laying eggs.



White comb: Ringworm, fowl pox or early stage of frostbite.



Dark red: poor circulation, stroke, heart Failure, lack of oxygen in blood, breathing Or respiratory issues.

Chicken Droppings

Chicken droppings are one of the most important things to check every time because you get to know whether your birds are healthy, or attacked by a disease. When you understand the type of droppings the chickens give out, you will be able to act immediately and prevent losses; You need to maintain a close eye on your birds.

Types of Droppings and what they could mean:

1. Firm greyish with a chalky white top.

This is a normal poo. It can be greyish, brownish, green or black, depending on what the chicken eats. If it's in solid state with the white solid part, the poo is very normal.

2. Watery diarrhoea

Could indicate high protein diets or kidney damage.

3. Clear or watery running droppings

Might be due to heat stress or, an infection with Infectious bronchitis.

4. Blood and mucous in stool

Coccidiosis

5. Yellow or green droppings with pasting of vent feathers

Likely caused by Fowl typhoid.

6. Greenish diarrhoea

Could be Newcastle disease.

7. Whitish pasty diarrhea

Infection with Infectious Bursal Disease (Gumboro disease).

8. White milky running stool

Pullorum disease

9. Yellow and foaming droppings

Worms or early signs of coccidiosis.

10. Brown diarrhea

Could be E. coli or Infectious bronchitis.

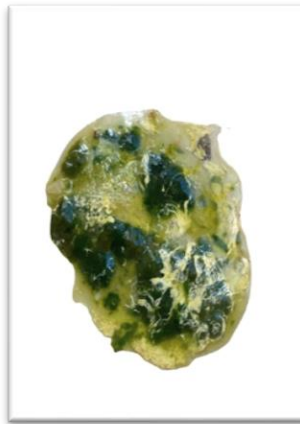
11. Worms in stool

Presence of internal parasites (worms).

Types of Droppings



1. Normal poo



2. Newcastle disease



3. Infectious bronchitis
Heat stress.



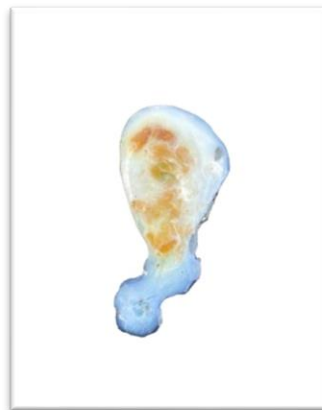
4. Coccidiosis.
Early sign



5. Coccidiosis



6. Necrotic enteritis



7. Pullorum disease



8. Gumboro disease

SOMALI INDIGENOUS CHICKENS

Somali indigenous chickens, also known as Wadani, are breeds that have adapted to the specific environmental conditions in parts of the Horn of Africa. These chickens are typically well-suited to the arid and semi-arid climates of the region, Digaag wadani are typically raised by small-scale farmers in the Somali region of Ethiopia. These chickens play an important role in the livelihoods of many rural households, providing a source of income, food security, and nutrition.

Characteristics of Somali indigenous chickens are:

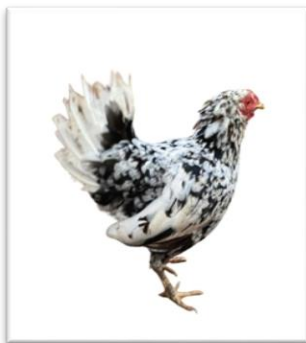
Hardiness: Somali indigenous chickens are known for their resilience and ability to thrive in harsh environmental conditions, including high temperatures, limited water availability, and scarcity of resources. They are characterized by slow growth, late maturity & low production performance; but resistance to certain diseases & parasites

Adaptability: These chickens are dual-purpose breeds and adapted to the local environment and are often able to forage for their own food, including insects, seeds, and vegetation. They are well-suited to free-range and semi-scavenging production systems.

Genetic Diversity: Somali indigenous chickens exhibit a range of genetic diversity, with variations in size, color, plumage patterns, and other traits. This diversity reflects the diverse agroecological zones found within region.

Types of Somali indigenous chickens

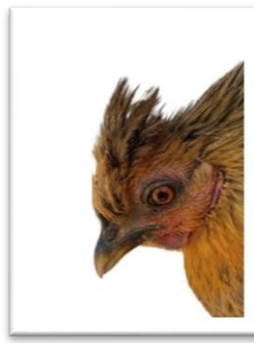
They are divided in to 4 groups:



1.SHUGURI



2. WADANI



3. SHINAAWI



4.CAGO BUL BUL

Names of Somali indigenous chickens



1. Digaagada madow



2. Diiq madow



3. Cado



4. Caynosh



5. Dhiin dhiin



6. Giir giir



7. Dhabar madow



8. Dhabar buni



9. Shuguriyad



10. shuguri



11. kur shaxan



12. Diiq cas



13. Buniya



14. Tiix



15. Cawlan



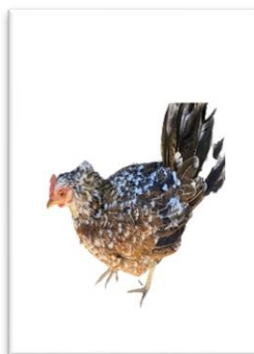
16. Dhabar daah



17. Boor



18. Car -Car



19. Bar-Bar



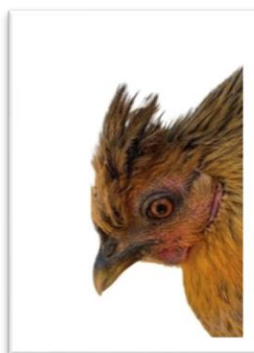
20. Hurdi



21. Sharaxan



22. Qoor dahab



23. Shinaawi



24. Bar dahab

REFERENCES

- Ahmed D. Anjum. (1996). poultry diseases diagnosis coloured atlas. Faisalabad: Arbi Publications.
- Drug administration and control authority of Ethiopia. (2006). Standard treatment of guidelines for veterinary practice. Adis ababa: Chamber printing house.
- FAO. (2010). Picture book of infectious poultry diseases. Southern Africa: Printing and Publishing Company Botswana PPCB.
- Gail Damerow. (2015). The chicken Health hand book (2nd ed.) North Adams: Storey publishing.
- Gordon and Jordan. (1982). Poultry diseases (2nd ed.) London: Bailliere Tindall.
- International Livestock Research Institute (ILRI). (2017). Guide to chicken health and management in Ethiopia.
- J.L. Vegad and A.K. Katiyar. (2015). A Text book of veterinary special pathology, infectious diseases of livestock and poultry. Delhi: Neekunj print process.
- J.L. Vegad. (2007). A colour atlas of poultry diseases. Delhi: International book distributing CO.
- Mohd. Osamah Kalim. (2012). Poultry diseases and their control. New Delhi: Enkay publishing house.
- MSD Animal health. Important poultry diseases
- Peter L. Msotte and Carol J. Cardona. (2009). Poultry disease handbook for Africa. Published by the Global Livestock Collaborative Research Support Program.
- Simon M. Shane and Professor Emeritus. (2005). Handbook on poultry diseases (2nd ed.) Singapore: American Soybean Association.
- Veterinary drug and animal feed administration and control authority of Ethiopia. (2020) Standard veterinary treatment of guidelines for Ethiopia (2nd ed.) Adis ababa, Ethiopia.

APPENDIX

Appendix 1. English-Somali terminology of poultry science

English	Af-Soomaali	English	Af-Soomaali
Abdomen	Ubuc	Comb	Dhoor
Anemia	Dhiigyari	Coughing	Qufac
Anti	Lid	Crop	Kiish
Artery	Halbawlaha	Culling	Xero kasaarka
Ascaris	Calooljilaabte	Depression	Niyad jab
Ataxia	Dhac dhac	Diaphragm	Bog
Avian Influenza	Hargabka shimbiraha	Diet deficiency	Nafaqadaro
Avian pox	Dhow, furuq shimbiraha	Duck	Booleanboolad
Biosucurity	Xayn daab	Duodenum	Ganac
Blind	Indhalaan	Emaciation	Wayd
Bone	Laf	Energy	Tamar
Botulism	Qoorjabiye	Excretion	Qashinsaarka
Broiler	Dhaylo	Eye inflammation	Indho xanuun
Cannibalism	is-cunka digaaga	Eyelid	Dahaarka isha
Capon	Doore	Family	Bah
Carrier	cudur-side	Fat	Baruur
Cataracts	Caad	Feaces	Saxaro
Chick	Jiiq-jiiqle	Feather	Baal
Chromosome	Hidho side	Feeding	Quudin
Chronic	Cudur dabadheeraada	Flea	Takfi
Class	Gacal	Fluid	Dareere
Cock	Diiq	Fly	Digsi
Cockerel	Qaan	gasp	Hiinraag
Colon	Malawad	Genus	Duul

English	Af-Soomaali	English	Af-Soomaali
Growth	Koriimo	Mosquito	Kaneeco
Guidelines	Hab-raac	Muscle	Muruq
Hackles	Daliig	Needle	Irbad
Helminthes	Dirxi calooled	Nest	Cush
Hen	Digaagad	Newcastle Disease	Qoor maroojiye
Hookworm	Gooryaan mulaaxeed	Oesophagus	Hunguriga cad
Huddling	Isku ururid	Ointment	Boomato
Hybrid	Iska dhal	Order	Dir
Hygiene	Nadaafada	Ovum	Ugxaan-side
Immune system	Habdhiska difaaca	paralysis	Qalal
Inhalation	Neefqaadasho	Pasteurization	Karkarin
Inheritance	Dhaxaltooyo	Phylum	Qayb
Injection	Muditaan	Pigeon	Xamaam/ Badeli
Insemination	Bacriminta	Pullet	Qaymad
Internal parasites	Caal	Pulse	Garac wadnaha
Isolation	Karantiimeyn	Pus	Malax
Joint	Xagal	Rectum	Malawad
Kingdom	Boqortooyo	Simple comb	Dhoor tag
Lameness	Dhutinta	Spent hen	Digaagad xafashtay
Larynx	Dalqo	Spur	Cidiyo xaraan
Liver	Beer	Trachea	Hunguri
Louse	Injir	Wattle	Kolamo
Mating	Saarasho	Zygote	Bacrin

Appendix 2. Differential diagnosis of poultry diseases

Sign	Torticollis	Paralysis	Sever paleness (Comb)
Diseases	1. Fowl cholera 2. Newcastle disease 3. Marek's diseases 4. Avian toxoplasmosis	1. Newcastle diseases 2. Botulism 3. Aflatoxin 4. A.E 5. Spirochetosis 6. Marek' diseases	1. Coccidiosis 2. Parasite infestation
Sign	Blindness	Conjunctivitis	Swollen head
Diseases	1. E. coli 2. Fowl pox 3. Aspergilosis 4. Paratyphoid 5. Marek's diseases 6. A. E	1. E. coli 2. Pox 3. Increase ammonia 4. Vit.A. deficiency	5. Infectious coryza 6. Newcastle disease 7. E. coli 8. TRT
Sign	Hemorrhage in Proventriculus	Caseated exudates	Neoplastic diseases
Diseases	1. Hemorrhage at papillae: Newcastle disease 2. Thickening of the wall: Marek's diseases 3. junction between proventriculus and gizzard. Gumboro disease	1. E.coli 2. F. Cholera 3. Vit A Defi.. 4. Staph spp 5. Aspergilosis 6. Mycoplasma	1. Marek's disease 2. Avian leucosis

Appendix 3. Vaccination schedule

Breed

Hatch no

Total no. of chicks.....

Date of hatch

	Age (days/weeks)	Vaccinate type	Way of administration	Remarks
1	Day-old	Mareks	Sub cutaneous	
2	Day 3	NCD/HB1/	Ocular	
3	Day 14-18	Gumboro	Ocular	With 7 days difference
4	Day 21-25	Gumboro	Water / ocular	
5	Day 27	NCD/Lasota/	Water	
6	Day.....45	Fowl typhoid	Sub cutaneous	
7	Day 63	NCD/Lasota/	Water	
8	Day 72	Fowl pox	Wing-web	
9	Day.....90	Fowl typhoid	Sub cutaneous	
10	Day.....112	NCD/Lasota/	Water	
11	*Week.....28	“	“	
12	*Week.....40	“	“	
13	*Week..... 52	“	“	
14	*Week.....64	“	“	
15	*Week.....76	“	“	
16	*Week.....88	“	“	

Note * After 112 days of age, NCD/Lasota/ is given every 12 weeks.

National poultry research case team (DZARC)

Appendix 4. Family bacteria of poultry

	Family	Grams	Diseases in poultry
1	<i>Staphylococcus aureus</i>	Positive	Bumble -foot
2	<i>Streptococci species</i>	Positive	Septicaemia and Endocarditis
3	<i>Mycobacterium avium</i>	Positive	Tuberculosis
4	<i>Clostridium septicum</i>	A, Positive	Necrotic dermatitis
5	<i>Clostridium colinum</i>	A, Positive	Ulcerative enteritis
6	<i>Clostridium perfringens</i>	A, Positive	Necrotic dermatitis and Sudden death
7	<i>Clostridium botulinum</i>	A, Positive	Botulism
8	<i>E. Coli</i>	Negative	Omphalitis-airsacculitis- peritonitis
9	<i>Salmonella pullorum</i>	Negative	Pullorum disease (bacillary white diarrhia)
10	<i>Salmonella gallinarum</i>	Negative	Fowl typhoid
11	<i>Salmonella typhimurium</i>	Negative	Fowl para typhoid-swollen joints-enteritis
12	<i>Pseudomonas species</i>	Negative	Omphalitis
13	<i>Pasturella multocida</i>	Negative	Fowl cholera- swollen joints
14	<i>Campylobacter jejuni</i>	Negative	Vibronic hepatitis
15	<i>H. paragallinarum</i>	Negative	Infectious coryza
16	<i>Chlamydia psittaci</i>	Negative	Psittacosis

NB: A, Positive means Anaerobic Positive.

Appendix 5. Summary of poultry diseases

Disease	Etiology	Signs	Postmortem	Diagnosis	Treatment	Vaccine/Control
New Castle (ND)	Paramyxo Type-I	Greenish Diarrhea Gasping Torticollis	Haemorrhages on tip of Proventriculus and Cecal tonsils	Signs Postmortem ELISA H.I	ND colon 1cc/2-6 L H ₂ O Aspirin 1cc/1L H ₂ O Novacoc 1cc/2-6L H ₂ O	NDV 100 ml 1st week eye drop 3rd week 0.5cc inj. 10th week 1cc inj. Every 2 month 1cc inj.
Infectious Bronchitis (I.B)	Corona Virus	Urate Plug Gasping Tracheal Rales Misshapen egg	Exudate in trachea and nasal cavity Caseous plug in bronchi Kidney pale swollen Salpingitis	Postmortem ELISA H.I AGPT DDx ND ILT A.I	E-sel 1cc/6L H ₂ O Aspirin 1cc/1L H ₂ O Novacoc 1cc/2-6L H ₂ O Flushing to remove pug	Avoid Overcrowding Biosecurity Vaccine
Avian Influenza (A.I)	Orthomyxo virus type A	Cyanosis of wattle and comb Ruffled feather Edema of head Diarrhea	Pin point haemorrhages on base of proventriculus Shank and on All viscera	Signs Postmortem DDx ND ILT I.B Mycoplasma Coryza	No treatment Supportive	Biosecurity Strict Quarantine All in and all out Vaccine
Infectious Laryngo-tracheitis (ILT)	Herpes virus	Sneezing Dyspnea Blood tinge nasal discharge Conjunctivitis	Tracheitis Blood in trachea Conjunctivitis	No response to treatment Blood tinge nasal discharge	No treatment Supportive	Biosecurity Strict Quarantine All in and all out Vaccine
CRD CCRD	Mycoplasma Myco + E.coli	Tracheal rale Drooling Sneezing Emaciation Slow FCR Lameness	Tracheitis Exudate in trachea Yellow colour cheesy flakes around lungs Salpingitis Synovitis Pericarditis Peri-hepatitis	Signs Postmortem	Tilmicosin 260mg/ml 1 ml / 12.2 kg	Purchase of uninfected chicks All in and all out Biosecurity Preventative medication
Infectious bursal disease Gumboro	Birna virus	Young birds Sudden mortality at 3-7 days Swollen/atrophy of bursa	Haemorrhages on thigh & musculature Atrophy of bursa Swollen, pale kidney	Signs Postmortem	No treatment Supportive	Control Stress Control dehydration Feed dilution Multi vitamins Flushing
Marek's Disease	Herpes virus	Tumor on feather Blindness Grey iris Paralysis	Grey white neoplastic lesions on liver spleen	Signs Postmortem	No treatment Supportive	Vaccine At 0 day in hatchery
Infectious Chicken Anemia	Gyro virus Circoviridae	Poor growth Pale birds Sudden mortality at 13-16 days	Pale bone marrow Atrophy of thymus & bursa Discoloured liver & kidney	Signs Postmortem	No treatment Supportive	Vaccine Live in breeder flock 6 weeks before incubation of eggs

Infectious Coryza	Haemophilus paragallinarum	Facial swelling Upper respiratory signs Swollen wattle Purulent ocular/nasal discharge	Catarrhal inflame. Of nasal sinus Conjunctivitis Haemorrhagic trachea	Signs Postmortem	Tylosin 10-20mg/kg Tylo + Doxy 10 : 20 1 g/2L H ₂ O	Avoid stress Prophylactically Tylo + Doxy 10 : 20 1 g/2L H ₂ O
Fowl Cholera	Pasteurella multocida	Swelling of neck Diarrhea Nasal discharge Coughing Swollen joints	Focal hepatitis Star shape white foci on liver Enteritis	Signs Postmortem DDx Strept aureus Coryza Mycoplasma	Sulpha drugs Penicillin Norfloxacin	Biosecurity Strict Quarantine All in and all out Avoid Rodents
Typhoid	Salmonella gallinarum Affect any age	Ruffled feathers Thirst Yellow diarrhea Reluctance to move.	Pale green enlarged liver with small necrotic foci & congestion Engorgement of kidney & spleen Enteritis of anterior small intestine	Signs Postmortem	Florfenicol 1cc/2 L H ₂ O	Biosecurity Strict Quarantine All in and all out
Pullorum Disease	Salmonella pullorum	Whitish diarrhea White fecal pasting on vent	Grey nodules in the liver, spleen, lungs, heart, gizzard, and intestine. Firm, cheesy material in the ceca.	Signs Postmortem	Florfenicol 1cc/2 L H ₂ O	Biosecurity Strict Quarantine All in and all out
Necrotic Enteritis	Clostridium perfringens	Closed eyes Immobility Golden yellowish diarrhoea	Small intestine (usually middle to distal) thickened and distended Golden yellow intestine	Signs Postmortem	Co-Amoxyclave Clavulanic acid + Amoxicillin	Penicillin in feed Probiotics Biosecurity Strict Quarantine All in and all out
Aspergillosis	Aspergillus fumigatus	Silent gasping Nervous signs (rare) Ocular discharge	Yellow to grey nodules or plaques in lungs, air sacs, trachea, peritoneal cavity, may have greenish surface. Conjunctivitis/keratitis.	Signs Postmortem	Amphotericin B Nystatin 1cc/1 L H ₂ O	Nystatin in feed Dry Litter Hygiene Good quality feed
HPS (Angara)	Adeno virus	Sudden increase in mortality Yellow mucoid droppings	Excessive straw-coloured fluid in pericardium (up to 10 ml) Enlarged, pale friable liver and kidney Lungs edematous	Signs Postmortem	Flushing Stralka 1cc/1 L 4h x 3 day	Biosecurity Strict Quarantine All in and all out

INDEX

Aflatoxin, 70
Ascaridiasis, 84
Aspergillosis, 76
Avian encephalomyelitis, 27
Candidiasis, 73
Cannibalism, 99
Capillariasis, 84
Chicken Mite, 93
Citrinin, 71
Colibacillosis, 41
Common Chigger, 93
Cyclopiazonic Acid, 71
Dermatophytosis, 80
Echidnophaga gallinacea, 89
Ergotism, 71
Feather loss, 107
Fowl cholera, 47
Fowl pox, 16
Fowl typhoid, 50
Fusariotoxicosis, 70
Gangrenous dermatitis, 44
Gastrointestinal parasitism, 82
Infectious bronchitis, 11
Infectious bursal disease, 24
Infectious coryza, 35
Infectious laryngotracheitis, 32
Lice, 93
Lymphoid leukosis, 30

Marek's disease, 7
Mycotoxicosis, 70
Necrotic enteritis, 56
Newcastle disease, 1
Ochratoxin, 70
Oosporein, 71
Prolapsed vent, 105
Pullorum disease, 53
Rickets, 95
Scaly leg mite infestation, 85
Staphylococcosis, 60
Vent gleet, 103
Vitamin A deficiency, 97
Vitamin D3 deficiency, 98
Vitamin E deficiency, 98

ATLAS OF POULTRY DISEASES

GUIDELINES FOR VETERINARIAN



Yahia Yasin Hassan was born in Chisimaio in 1991. He earned his Diploma in Pharmacy in 2012 from Eastern Africa International College. In 2019, he obtained his Doctor of Veterinary Medicine from the College of Veterinary Medicine at Jigjiga University. Additionally, he completed his MSc in One Health in Tropical and Infectious Diseases in 2021 from the Department of Public Health at the College of Medicine and Health Sciences, Jigjiga University.

Yahia Yasin Hassan kick started his career as an animal health officer in the Harorays district. He then transitioned to working as an assessor of animal health at the Agency of Competence in the SRS. Adding to his professional experience.

He served as a lecturer at the College of Veterinary Medicine at University of Kebri Dehar. Yahia Yasin Hassan has a prolific portfolio, having published research papers and authored four books. Currently, he is founder of Aaran Poultry Consultancy, a leading firm specializing in poultry health management, consultancy, and training services.

----Dr. Yahia Y. Hassan

Poultry Farm Practitioner | Poultry Pathologist | Poultry Expert

Atlas of poultry diseases is a comprehensive visual guide that illustrates various diseases commonly affecting poultry. This book provides detailed information on the symptoms, diagnosis, treatment, postmortem examination, prevetion and control of poultry diseases, making it an essential reference for poultry farmers, veterinarians, and anyone involved in poultry industry.